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DISEASES OF THE HEART.



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DISEASES OF THE HEART

AND

THORACIC AORTA.

BY

BYROM BRAMWELL, M.D., F.R.C.P.E.,

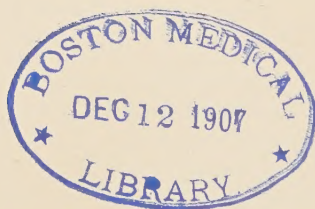
LECTURER ON THE PRINCIPLES AND PRACTICE OF MEDICINE, AND ON PRACTICAL MEDICINE AND
MEDICAL DIAGNOSIS IN THE EXTRA-ACADEMICAL SCHOOL OF MEDICINE, EDINBURGH; PATHO-
LOGIST TO THE EDINBURGH ROYAL INFIRMARY; ADDITIONAL EXAMINER IN CLINICAL
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HOME, THE TYNE FLOATING HOSPITAL,
ETC., ETC.

WITH 317 ILLUSTRATIONS.

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In Memoriam.

J. B. B.

PREFACE.

IN the following pages, which are based on portions of my Lectures on the Principles and Practice of Medicine, and on Practical Medicine and Medical Diagnosis, I have endeavoured to give a systematic account of the Diseases of the Heart and Thoracic Aorta.¹

My attention was early directed to the diseases of the heart, for soon after entering practice I met with a long series of rare and interesting cardiac cases. During the past fifteen years I have been constantly thinking and talking about the subject, and steadily accumulating the clinical knowledge and pathological material necessary for the production of such a work.

The lithographs of naked-eye objects, represent with few exceptions the hearts of patients who have been under my own care during life, and with whose clinical histories I am intimately acquainted. The microscopical lithographs are, with two exceptions, copied from sections made by myself. In order to ensure absolute accuracy of representation, the naked-eye specimens were first photographed and then drawn under my immediate personal supervision, while the microscopical objects have been placed directly on the stone from my own drawings.

¹ The subject matter of the work was delivered almost exactly as it stands, in the form of lectures to the author's class at the beginning of the winter session 1883-84.

I am particularly indebted to Professor Turner for his kindness and liberality in allowing me to photograph and reproduce the rare and beautiful specimens which are shown in figs. 170', 171, 242, 243, 244, 262, and 281, and which are contained in the Anatomical Museum of the University of Edinburgh. I am also indebted to Professor Greenfield and Dr Wyllie, with whose kind consent two specimens (figs 169 and 263) which came under my notice in the course of my work as Pathologist to the Edinburgh Royal Infirmary, are represented.

My thanks are also due to Professor Pettigrew, Drs Walshe, Sansom, Galabin, Fothergill, Green, Macalister, Dudgeon, and The Executors of the late Dr Peacock, for their kindness in allowing me to reproduce some of the figures which have appeared in their respective works; and to Messrs J. and A. Churchill, Messrs Longmans, Green and Co., and Messrs Macmillan and Co., for permitting me to copy, or to have electrotypes of cuts, from works which they publish.

I must also express my indebtedness to the numerous writers whose opinions are referred to in the text, and more especially to Drs Walshe, Sibson, Sansom, and Galabin.

B. B.

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DISEASES OF THE HEART.

CHAPTER I.

INTRODUCTORY ANATOMICAL AND PHYSIOLOGICAL REMARKS THE ARTERIAL BLOOD-SUPPLY OF THE HEART. THE AUTOMATIC MECHANISM OF THE HEART. THE NERVOUS SUPPLY OF THE HEART. THE CONNECTIONS OF THE SYMPATHETIC WITH THE HEART. THE CONNECTIONS OF THE PNEUMOGASTRIC WITH THE HEART. THE MANNER IN WHICH THE VAGUS AND SYMPATHETIC NERVES AFFECT THE HEART. THE RELATIONSHIP BETWEEN THE HEART AND THE MINUTE BLOOD-VESSELS

THE consideration of the Diseases of the Circulatory System is a subject of great importance, for it embraces many affections which are of every day occurrence in practice, which entail a vast amount of suffering, and which are very frequently the cause of death.

The diseases of the Circulatory System include¹—

1. The diseases of the Heart and Pericardium.
2. The diseases of the Arteries.
3. The diseases of the Veins.

In treating of the Diseases of the Heart and Pericardium, I shall first describe the methods of clinical examination which are in common use for investigating the condition of these structures, directing attention, as I proceed, to those

¹ The diseases of the Lymphatics are sometimes included under this head, but they are, I think, more appropriately considered under the disorders of the Chylopoietic Viscera.

points in their anatomy, physiology, and pathology, which are essential for the due comprehension of our subject. After the reader has become thoroughly familiar with these points, I shall consider the individual affections of the heart and pericardium in detail.

PRELIMINARY ANATOMICAL AND PHYSIOLOGICAL CONSIDERATIONS.

The heart—the central organ of the circulation—may be regarded as a muscular pump; or, speaking more accurately, it may be said to consist of two¹ muscular pumps,—the systemic and pulmonary hearts respectively.

Each half or pump consists of two chambers, viz., (1) a *receiving* chamber or *auricle*, and (2) a *propelling* chamber or *ventricle*.

¹ At an early period of foetal life, as in the permanent state of the Dugong (see fig. 1), the heart is so deeply cleft from the apex towards the base, as almost to give the idea of two separate organs.—*Carpenter's Physiology*, p. 271. In the adult heart of man, the remains of the deep cleft are sometimes seen in the form of a bifid apex (see fig. 2).

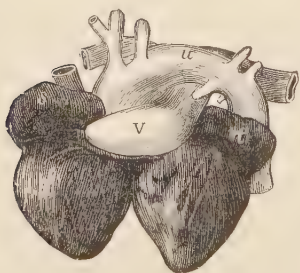


FIG. 1.



FIG. 2.

FIG. 1.—Heart of Dugong seen anteriorly: shows bifid apex. *s*, right auricle. *g*, right ventricle. *t*, left auricle. *r*, left ventricle. *u*, aorta giving off innominate, left carotid and left subclavian arteries. *v*, pulmonary artery bifurcating and proceeding to right and left lungs.—(After Owen.)

FIG. 2. Apex of the human heart (half the natural size) showing a deep cleft, A, between the two ventricles.

The function of the auricle is to receive the blood which is carried to it by the great veins, to store up that blood for a brief period (*i.e.* the period corresponding to the ventricular systole), and to transmit it to the ventricle. The function of the ventricle is to propel the blood, which it receives from the auricle, into the great artery which arises from it, and thence through the arterial system and round the vascular circle.¹

The backward flow of blood from the ventricle to the auricle is partly prevented by the auriculo-ventricular valve segments, partly, as Ludwig, Hesse, and Macalister have shown, by the contraction of the muscular wall of the ventricle itself, *i.e.* by the contraction of the muscular fibres which surround the valvular orifice. The backward flow from the arterial system (aorta or pulmonary artery) into the ventricle, is prevented by certain valvular arrangements, which I shall afterwards describe in detail; while the backward flow of blood from the auricles to the veins is partly prevented by the contraction of the muscular fibres, which are placed at their points of termination, *i.e.* where the veins join the heart, the systemic venous circulation being still further protected from the 'backwash,' which not unfrequently occurs through the tricuspid, even in conditions of health, by the valves of the veins themselves. By these means the onward flow of blood in one—a forward—direction is accurately maintained.

The course of the circulation is diagrammatically represented in figure 3, while the passage of the blood through the heart takes place in the following manner:—

At the commencement of the auricular diastole (and while the ventricular systole is taking place) blood begins to flow from the great venous trunks into the auricular

¹ The chief cause, of the motion of the blood, is the heart, but the onward passage of blood is also aided by the contraction of the blood vessels. This contraction is partly the result of elasticity, and partly due to an active contraction of the muscular coat.

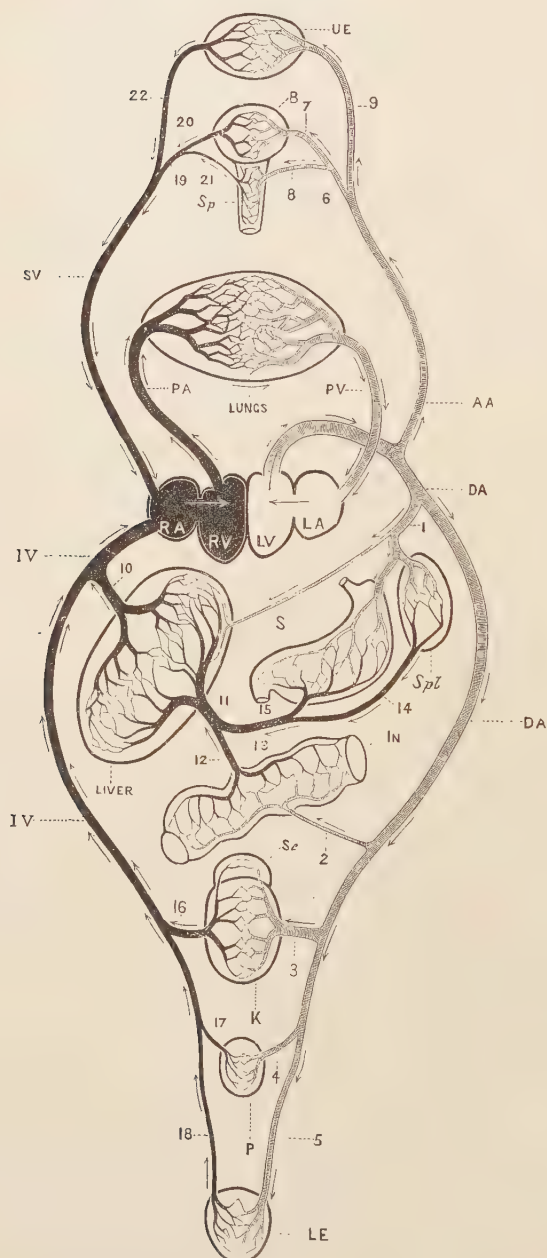


FIG. 3.

*Description of FIG. 3.—Diagrammatic representation of the course of the circulation.
(Modified from Dalton.)*

The arrows indicate the course of the circulation.

RV, the right, and LV, the left ventricles.

RA, the right, and LA, the left auricles.

Lungs, Liver (description in full).

S, Stomach.

Spl, Spleen.

In, Intestines.

K, Kidneys.

Sc, Supra-renal capsules.

P, Pelvic viscera.

LE, Lower extremities.

UE, Upper extremities and superficial parts of the head and neck.

B, Brain.

Sp, Spinal cord.

PA, Pulmonary artery.

PV, Pulmonary veins.

AA, Branches of the ascending aorta.

DA, Descending aorta.

SV, Superior cava.

IV, Inferior cava.

1 Cæliac axis, sending branches to the spleen, stomach, and liver.

2 Mesenteric arteries.

3 Renal arteries.

4 Arteries to the pelvic viscera.

5 Arteries to the lower extremities.

6 Arteries to brain and spinal cord.

7 Arteries to brain.

8 Arteries to spinal cord.

9 Arteries to the upper extremities and superficial parts of the head and neck.

10 Hepatic veins.

11 Portal vein.

12 Veins from the intestine (superior and inferior mesenteric veins).

13 Veins from the stomach and spleen.

14 Splenic vein.

15 Coronary and pyloric veins from the stomach.

16 Renal veins.

17 Veins from the pelvic viscera.

18 Veins of the lower extremities.

19 Veins from the brain and spinal cord.

20 Veins from the brain.

21 Veins from the spinal cord.

22 Veins from the upper extremities and superficial parts of the head and neck.

cavities; as soon as the ventricular diastole occurs the ventricles dilate, the pressure in the ventricular cavities becomes negative, and blood flows from the auricles into the ventricles through the auriculo-ventricular (mitral and tricuspid) orifices; before long the auricles become full of blood, and the auricular systole occurs; the muscular contraction commences in the walls of the great veins, and spreads in a peristaltic manner over the auricles, which sharply contract and discharge their blood into the ventricles, distending them, and bringing the segments of the auriculo-ventricular valves into close apposition; the muscular contraction now passes to the ventricles, the auriculo-ventricular orifices are firmly closed by the muscular contraction, to which I have already referred, the valve segments are tensely stretched, the arterial valves are burst open, and the ventricles empty themselves into the aorta and pulmonary artery; the great arteries become distended, the segments of the aortic and pulmonary valves are floated together; the recoil of the aorta and pulmonary artery then occurs, and the aortic and pulmonary valves are closed and stretched.

Now in studying both the physiology and pathology of the heart, and in investigating its diseases at the bedside, it is essential to keep this fact constantly in view, *that the heart is not merely a mechanical, but that it is also a muscular pump*, and that, as Professor Michael Foster so lucidly points out, its action consequently presents problems which are *partly mechanical and partly vital*.¹ I cannot insist too strongly upon the importance of looking at the heart both as a mechanical pump and as a vital organ; it is in fact the base upon which an intelligent comprehension of the physiology and pathology of the heart, and of the symptomatology, diagnosis and treatment of its diseases, must be founded.

But although the heart is a muscular organ, it differs from the other muscles of the body in several important particulars. They are as follows:—

¹ *A Text Book of Physiology*, p. 135.

In the *first* place, although the cardiac muscle is involuntary in its action, it is transversely striated in its structure—a fact which, as Gaskell has pointed out, explains many of the peculiarities of the cardiac muscle.

Dr Gaskell looks upon the heart as a specially modified portion of the vascular system; ‘the heart is to be considered,’ he says, ‘as a piece of artery or vein, the muscular walls of which have developed in a special manner. The keynote therefore of the peculiarities of the cardiac muscle consists in its structural position, intermediate between unstriated and striated muscle fibre. Muscular tissues exhibit three different modes of responding to stimulation according to their structure. These modes may be expressed by saying that certain muscles possess essentially the power of “tonic contraction,” others the power of “rhythmical contraction,” and others that of “rapid contraction.” A comparison of the tetanising action of a strong interrupted current upon a strip of muscle from the bladder of the tortoise and from the heart of the tortoise with the ordinary tetanus curve of the frog’s gastrocnemius shows clearly the difference of the three kinds of muscular tissue.

‘The unstriated muscle of the bladder contracts slowly after a long latent period, the contraction increasing steadily in force during and even after the cessation of the tetanizing current, and then the strip returns with excessive slowness to its original length. In other words, we see a prolonged tonic contraction as the result of the stimulation.

‘With the striated muscle we have the well known curve of tetanus composed of the superposition of a series of rapid contractions.

‘The cardiac strip gives a curve which is intermediate between the two, and may be described as consisting of a long continued tonic contraction upon which a number of rapid contractions are superimposed. These separate rapid contractions never succeed one another so quickly as to fuse together. The cardiac muscle then, when tetanized, gives, in virtue of its relationship to unstriated muscle, a tetanus of tonicity (to use Ranvier’s expression), and at the same time a series of rapid contractions in consequence of its affinity to ordinary striated muscle. When the vitality of the tissue is impaired by exhaustion, by injury, by malnutrition, the cardiac muscle loses its power of rapid contraction, and the less-specialized tonic power alone remains, the muscle strip tetanized when in that condition contracts with a prolonged tonic contraction in the same way as unstriated muscle.

‘In another respect too, the intermediate position of the cardiac muscle between the slowly contracting, slowly exhausted unstriated muscle, and the rapidly contracting, easily exhausted striated muscle, is clearly shown; the vitality of unstriated muscle after the death of an animal is wonderfully long; the irritability of the cardiac muscle after death is

less than this, but decidedly greater than that of the ordinary striated muscles.¹

Secondly, The structure of its fibres is somewhat different from the structure of the voluntary muscles.

(a) The fibres of the heart are made up of quadrangular portions (see fig. 4), each of which contains a nucleus, and each of which may, therefore, be regarded as a distinct muscle cell.

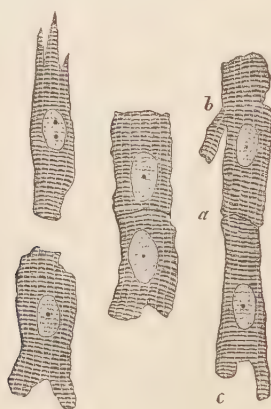


FIG. 4.—Six muscular fibre-cells from the heart, magnified 425 diameters.

(After E.A.S., Quain's *Anatomy*, Ninth Edition).

a, line of junction between two cells; b,c, branching of cells. (From a drawing by Mr J. E. Neale.)

(b) They have no sarcolemma.

(c) They are longitudinally as well as transversely striated, and, in good sections, are seen to be composed of a number of minute rods or fibres running parallel to each other.

(d) They freely anastomose, the connections being formed

¹ *The Journal of Physiology*, vol. iv., No. 2, p. 116.

by branches given off here and there, from the muscle cells of which the fibres are composed. (See fig. 5.)



FIG. 5.—Muscular fibres from the heart, magnified, showing their cross striæ, divisions, and junctions. (After Schweigger-Seidel, from Quain's *Anatomy*, Ninth Edition.)

The nuclei and cell-junctions are only represented on the right-hand side of the figure.

(e) They seem to be differently affected by electricity, the Faradic current, according to Ziemssen's observations, being much less efficacious in producing contractions and alterations in rhythm than the galvanic.

Thirdly, its action is constant, and its contractions rhythmic and automatic.

Amongst the vital problems connected with the heart, some of the most prominent and important are:—

(1) The manner in which it receives its arterial blood supply.

(2) The construction and mode of action of the mechanism, by which its contractions are produced and regulated (its

automatic muscular and *motor* nerve arrangements), the sensorium is informed of its condition (its *sensory* nerve arrangements), the nutritive condition of its muscular fibre is maintained and regulated (its *trophic* nerve supply).

(3) The construction and nature of the nerve arrangements, by means of which it is brought into relationship with the other parts of the vascular system, and with the other organs of the body.

The Arterial Blood-supply of the Heart.

As we all know, arterial blood is conveyed to the cardiac muscle by the coronary arteries; and until quite recently it was supposed by many of our leading physiologists and physicians, that in consequence of the relative positions of the parts, the orifices of the coronary arteries must of necessity be closed during the systole of the ventricle—the valve flaps being pressed against the orifices of the coronary arteries by the blood-stream in its passage from the ventricle into the aorta. The recent experiments, however, of Martin and Sedgwick, seem conclusively to show that this supposed closure does not occur, and that the coronary, like all the other arteries of the body, are distended during the systole of the heart. These observers have shown, by means of careful cardiographic tracings, that the blood-waves in the coronary arteries and carotids are exactly synchronous both in normal and diseased states of the circulation. It seems certain, therefore, as Dr George Balfour and others had previously argued, that the blood is propelled into the coronary arteries during the systole of the heart.

The Automatic Mechanism of the Heart.

The nervous mechanism of the heart is extremely complicated, and the manner in which it acts is far from being perfectly understood. It is a subject, however, of the greatest importance, and we must, therefore, consider it in some detail.

In the *first place*, it is necessary to remember that the contraction of the heart is automatic, *i.e.* it is due to impulses arising within the heart itself, and that, in conditions of health, the action of the two sides of the heart, *i.e.* of the two pumps, is synchronous. Under ordinary circumstances the heart beats in a perfectly rhythmical and regular manner, the number of contractions being, in the adult male, about 72 per minute.

Until recently it was supposed that the rhythmical action of the heart was entirely due to the periodical and orderly discharge of motor nerve force in the nerve ganglia, which are scattered through the organ, but recent observations, more especially of some German physiologists and the brilliant researches of Gaskell, seem to show that the influence of the cardiac ganglia is not indispensable, and that the muscular fibre itself, in some of the lower animals at all events, possesses the power of rhythmical contraction. Gaskell's observations seem to prove conclusively, that, in the tortoise, the automatic action of the heart does not depend upon any special rhythmical nervous apparatus, but that it is due to a property of rhythmical contraction inherent in the muscular tissue itself.

It is perhaps premature to conclude that the automatic contractions of the human heart are produced in exactly the same manner as the automatic contractions of the heart of the tortoise. We may, however, safely say, that in man, the rhythmical action of the heart must be due, either to the periodical and orderly discharge of motor nerve force from the ganglia which it contains, or to a rhythmical property possessed by the muscular tissue independently of any special nervous mechanism; and if we may judge by analogy, the latter view is possibly the correct one. And this we may term the *first step* in the comprehension of the mechanism of these complicated arrangements.

In the *second place*, it would appear that the stimulus to muscular contraction is the presence of blood, or rather the presence of blood under a certain pressure, in the cardiac

cavities.¹ If the pressure is too low the stimulus is insufficient, and the muscle does not contract. If, on the other hand, the pressure is too great, over-distention and a paralytic condition may result. Indeed the recently published experiments of Sewall and Donaldson seem to prove 'that, within its working limits of internal pressure, the heart muscle has a remarkable power of accommodating the intensity of its discharges of energy, to the resistance to be overcome.'²

'A fact continually forced upon our attention,' say these writers, 'was the great dependence of the systole, as to its energy and completeness, upon the amount of fluid within the heart. Particularly was this noticed in regard to the thin walled sinus and auricles, when isolated in the manner to be described. Within tolerably low fluid pressures, the sinus and auricles become so distended as to be powerless to contract; as the pressure within them is reduced they contract feebly, and when the quantity of blood flowing into them is only just sufficient to bring about full distension during diastole, they contract powerfully, and empty themselves completely at each beat. As the supply of blood is further lessened by lowering the pressure flask, the contractions apparently again become feebler, and in the sinus hardly visible.'³

The exact manner in which the cardiac muscle is stimulated by the presence of blood under a certain pressure is obscure. If the muscular contraction can occur independently of the ganglia, we must suppose either that the muscular fibre is *directly* stimulated by the mechanical stretching which it undergoes when the cardiac cavities become distended; or, that the contraction is indirect, and is due to stimulation of the fine nerve fibres⁴ which, as Schweigger-Seidel⁵ has shown, form a rich plexus in the endocardium. If we grant that the ganglia are concerned in the process, we may suppose, as some physiologists have for long held, that the mechanism is a reflex one. A cardiac ganglion-cell may be regarded as the centre of a reflex arc, to which a sensory nerve fibre passes from the endocardium, and from which a motor nerve

¹ In some of the lower animals, the frog for instance, the heart continues to beat even after the cavities have been cleared of blood, and indeed when they are almost empty of fluid. In the frog, therefore, the presence of blood is not absolutely necessary to produce cardiac contractions, but it is nevertheless probable that the pressure of the blood in the cardiac cavities, under ordinary circumstances, acts as a stimulus, and excites the contraction of the cardiac muscular fibres.

² *The Journal of Physiology*, vol. iii. p. 361.

³ *Loc. cit.* p. 361.

⁴ This view necessarily supposes that these fine nerve fibres are motor.

⁵ Quoted by Power, *Carpenter's Physiology*, p. 276.

fibre proceeds to the cardiac muscle (see fig. 6); the reflex mechanism being thrown into action by the presence of blood, under a certain pressure, in the cavities of the organ.

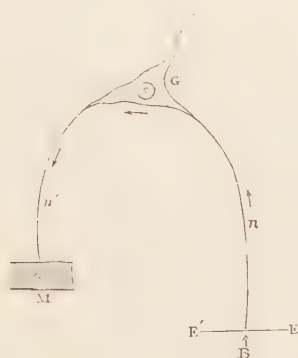


FIG. 6.—Diagrammatic representation of the reflex mechanism by which (it has been theorised) the cardiac muscle is thrown into contraction, under the influence of the blood pressure.

E', E, the endocardium; B, the blood in the cardiac cavity; M, the muscular fibre of the heart; G, ganglion cell, the reflex centre; n, sensory nerve fibre conducting the impression generated by the blood pressure on the sensory nerve terminations under the endocardium to the reflex centre; n', motor nerve fibril conducting the impulse from the reflex centre to the muscle.

In the *third place*, the experiments of Gaskell, and of Sewall and Donaldson, seem to show that in the frog and tortoise at least, and when the heart is acting normally, the motor impulse originates in the sinus, and passes from the sinus to the muscular fibres of the auricle, and thence to the ventricle, the rhythm of the sinus determining that of the whole heart.

Those authorities who believe that the automatic action of the heart is due to the periodical discharge of its motor ganglia, necessarily conclude, in order to explain these results, that the action of the ganglia, which are situated in the sinus venosus, is more powerful than that of the ganglia situated in other parts of the organ; and indeed some authorities have supposed that the ganglia of the sinus venosus are the true automatic centres, while the action of the other ganglia requires to be excited reflexly, or in some other manner. The supporters of the neurotic theory further believe that the sequence of the cardiac contractions (the fact that the contraction of the

auricle follows at a regular interval upon the contraction of the sinus, and the contraction of the ventricle at a regular interval upon that of the auricle) is due to nervous influences.

Dr Gaskell, whose experiments seem to show, as I have already mentioned, that the automatic rhythm originates in the muscular tissue itself, explains the fact that the contractions originate in the sinus, by the peculiar structure of the cardiac muscle in this part of the heart; while he states that the sequence of the contraction of the ventricle to the auricle is not due to separate stimuli passing along nerve fibres from the sinus to the ventricle—not, in short, to any nervous mechanism—but to the fact that a wave of contraction passes directly from the muscular fibres of the auricle to the muscular fibres of the ventricle, through the muscular fibres forming the auriculo-ventricular groove. The pause, or, more correctly, the alteration of rate in the progress of the contraction wave, which takes place between the contraction of the auricle and the contraction of the ventricle, is, he says, due to an alteration in the conducting power, which naturally exists at the auriculo-ventricular ring. ‘This diminished conducting power or natural block, exists not only because the auriculo-ventricular muscular ring is narrow, and that a somewhat abrupt change occurs in the direction of the muscular fibres along which the contraction wave passes when it reaches and leaves this ring, but essentially because the structure of the muscular fibres here is different from those of the auricle or ventricle.

‘The muscle fibres throughout the heart¹ are,’ he states, ‘of the same type, any differences which are seen are differences in the prominence of the various structural peculiarities of the cardiac type of muscle, and are not so great as the differences between unstriated and striated muscle fibres. Thus all the muscle fibres are, to a greater or less extent, transversely striated; but the prominence of this striation varies considerably. Similarly the thickness of the fibre, the extent of parallelism of its edges, the size of the nucleus in relation to the size of the fibre, and therefore the extent of crowding

¹ He is speaking, it must be remembered, of the adult heart of the tortoise.

of the nuclei in any strip, all present differences in different parts of the heart. The greatest contrast is seen when the muscular fibres of the ground layer in the sinus are teased out and compared with the muscle fibres of the spongy tissue of the ventricle. The sinus muscle fibre is thin and delicate, tapering somewhat at both ends, with a large central oval nucleus which causes a distinct bulging of the fibre; the substance of the fibre shows a striation which is decidedly indistinct, presenting often a granular rather than a distinct banded appearance. On the other hand, the ventricular muscle fibre is boldly and strongly striated, it is much thicker than that of the sinus, its edges are parallel, and the thin elongated nucleus is small in comparison to the size of the fibre. The muscle fibres of the reticulated tissue of the auricle are not so large or so coarsely striated as those of the ventricle, though larger and much more distinctly striated than the sinus muscle fibres. Their edges also are more parallel than in the fibres of the sinus. The muscular ring forming the junction of the auricles and ventricle, and, to a certain extent, the whole junction wall joining the two auricles, are composed of muscle fibres, with a structure intermediate between the sinus and the auricle muscles. The nuclei are large, conspicuous on section both in size and number, the striation is not so well marked as in the bulged portion of the auricles, and the fibres are thin and delicate, with somewhat parallel edges.

‘Such a structure as above described is very suggestive, not only as an explanation of the pauses which occur naturally in the course of the peristaltic wave of contraction, but also of the differences of rhythmical power exhibited by different parts of the heart.’¹

Dr Gaskell, therefore, conceives ‘that the variations in the rhythmical power and in conductivity, which are characteristic of the different parts of the adult heart of the tortoise, may all be accounted for on the supposition, that the development of the muscular tissue of the originally tubular heart has not proceeded at the same rate throughout the tube, so that in the adult heart greater variations in rhythmical power are

¹ *Journal of Physiology*, vol. iv. pp. 72, 73.

apparent in the different sections of it, than in the original tubular heart; the peristaltic wave of contraction which originally passed smoothly from end to end, passes finally along a tube of irregular calibre, the muscular walls of which have become so modified in their rates of contraction and conduction, as well as in the arrangement of the fibres, as to form out of a simple peristaltically contracting tube such an efficient muscular pump as is represented by the adult heart.¹

‘The conception advanced above, that the rhythmical beating of the heart is due to a series of peristaltic contractions which start from that particular portion of the muscular tissue of the heart in which the property of automatic rhythm has been most largely developed, brings the heart’s action into harmony with the rest of the vascular system and with the rhythmical properties which are so often manifested by the less specialized forms of muscular tissue.’²

Space will not allow me to detail the facts and arguments on which Dr Gaskell founds this theory, which obviously has most important practical bearings on the pathology of the human heart. I would, however, strongly advise my readers to peruse the original for themselves.

In the *fourth place*, Gaskell has distinctly proved that the ventricle is supplied with afferent nerve fibres, which are ‘able to regulate the force of the auricular contractions, as well as in all probability the rate of rhythm. It is then conceivable that the function of many of the nerve fibres which pass into the ventricle is by their action upon the force of the auricular contractions to regulate the amount of blood thrown into the ventricle, and therefore the amount of work done by the heart.’³

But although the movements of the heart are automatic, it is intimately connected both with the sympathetic and cerebro-spinal nervous centres; and its action can, as each one of us so well knows, be readily affected by general nervous influences. But in order that this most important and difficult part of our subject may, if possible, be clearly understood, I must now describe the nervous supply of the heart in some detail.

¹ *Journal of Physiology*, vol. iv. p. 77. ² *Loc. cit.* p. 80. ³ *Loc. cit.* p. 92.

Delicate nerve fibres are met with in considerable numbers both on the surface and in the substance of the heart; and nodular enlargements, which microscopic examination has shown to be true nervous ganglia, *i.e.* to contain nerve cells, are abundantly distributed on certain of these nerve fibres, and on the numerous points of junction which certain of the fibres make with each other. In the human subject the nerves of the heart are neither so numerous nor so distinct as in some of the lower animals, as, for example, the calf (see figs. 7 and 8); but even in man 'the surface and substance of the heart are enveloped in a more or less uniform plexus.' (*The Physiology of the Circulation*, by J. Bell Pettigrew, p. 298.)

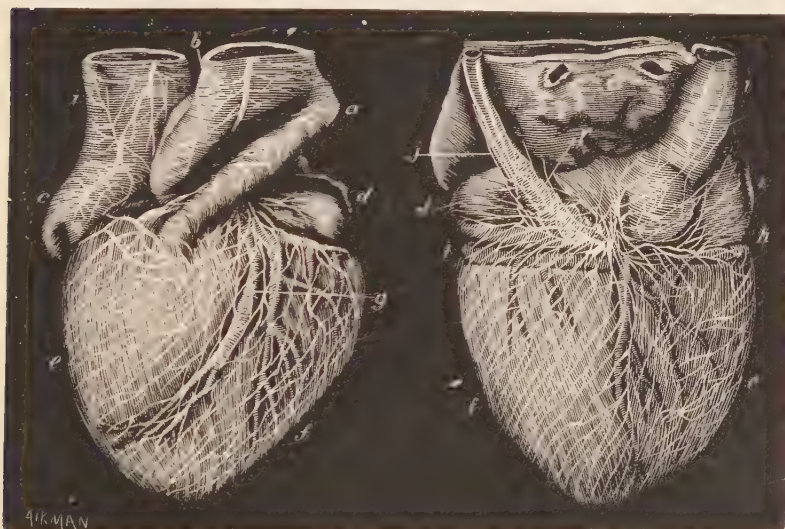


FIG. 7.

FIG. 8.

FIG. 7.—Nerves and ganglia on the anterior surface of the calf's heart. (After Pettigrew.)

a, b, pulmonary artery and aorta with nerve-plexuses and ganglia. *i*, descending cava with nerve-plexus and ganglia. *c*, right auricle. *d*, left auricle. *e*, Nerves and ganglia distributed on right side of heart. *f*, ditto on left side. *g*, anterior coronary vessels covered with nerve-plexuses and ganglia.

FIG. 8.—Nerves and ganglia on the posterior surface of the calf's heart. (After Pettigrew.)

i, descending cava. *c*, nerves and ganglia on right auricle. *d*, ditto on left. *e*, nerves and ganglia on right ventricle. *f*, ditto on left. *j*, great nerve-plexus and ganglia covering coronary sinus (*r*) and extending itself on the right (*h*), left (*l*), and posterior coronary vessels, and the right (*e*) and left (*f*) ventricles generally. The ganglia in this case are very numerous, particularly on the coronary sinus (*r*).

Both nerve fibres and nerve cells are most numerous and most distinct in the grooves which contain the larger branches of the coronary arteries, and anatomists have consequently described a *right* and a *left coronary plexus*.

The *coronary plexuses* (see fig. 9) may be said to be composed of branches proceeding to the heart from the so-called *cardiac plexus*, which, to a large extent at least, surrounds the arch of the aorta.

The *right coronary plexus* (R C P) accompanies the branches of the right coronary artery, and sends branches (a, b) both to the right auricle and right ventricle. It receives branches (c, d) from both the *superficial* (S C P) and *deep* (D C P) *cardiac plexuses* (see fig. 10) the composition of which I shall presently describe.

The *left coronary plexus* (L C P), which is larger than the right, is composed of two primary divisions (represented as one ganglion cell in the figure), corresponding to the primary divisions of the left coronary artery. The branches proceeding to it (e) are almost entirely derived from the left half of the deep cardiac plexus (D'C'P'), a few filaments only (f) passing to it from the right half (DPC). The branches proceeding from it (g, h, i) are distributed to the left auricle and to both ventricles.

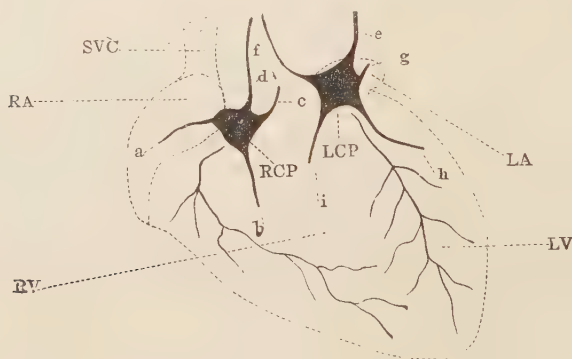


FIG. 9.—*Diagrammatic representation of the coronary plexuses.*

RCP, right, and LCP, left coronary plexuses; RV, right, and LV, left ventricles of the heart; RA, right, and LA, left auricles; SVC, superior vena cava. a, branch of the right coronary plexus to the right auricle; b, to the right ventricle; c and d, branches proceeding to the right coronary plexus from the superficial, and deep cardiac plexuses respectively; e and f, branches proceeding to the left coronary plexus from the left and right halves of the deep cardiac plexus respectively; g, branch proceeding from the left coronary plexus to the left auricle; h, to the left ventricle; i, to the right ventricle.

The *cardiac plexus* (see fig. 10, S C P, D C P and D'C'P') may be described as partly surrounding the arch of the aorta. In it all the cardiac branches of the sympathetic and of the pneumogastric and its branches, terminate, and from it, branches proceed to the heart. In this great cardiac plexus the numerous nerve filaments, proceeding to and from the heart, are re-arranged and re-distributed, and by this means the heart is brought into most intimate connection with many distant parts—a connection which the practical physician should always keep in view, for it explains many of the symptoms (apparently unconnected with the heart) which are met with in association with cardiac disease.

The *cardiac plexus* has been artificially divided by anatomists into two parts, which are respectively termed the *superficial* and *deep cardiac plexuses*.

The *superficial cardiac plexus* (S C P) lies for the most part in the concavity of the aortic arch. In it the *first*, or superficial cardiac nerve of the left side (1'C'N'), and the lower cervical branch of the left pneumogastric (8', fig. 10) terminate; and from it branches (c) proceed to the right coronary plexus. It also gives a few small filaments (j and k) to the pulmonary artery and anterior pulmonary plexus of the left side.

The *deep cardiac plexus* (D C P and D' C' P'), which is considerably larger than the *superficial*, is situated 'behind the arch of the aorta, between it and the trachea, and above the bifurcation of the pulmonary artery.'¹

In it, all the branches of the sympathetic and pneumogastric proceeding to the heart, except the first or superficial cardiac branch of the left sympathetic and the lower cervical branch of the left pneumo-gastric, terminate.

The deep cardiac plexus is described as consisting of two halves (right and left).

The *right half* (D C P) sends branches to—

- (1.) The right coronary plexus (d).
- (2.) The right auricle (l).
- (3.) The left coronary plexus (a few filaments) (f).

The great majority of the branches of the *left half* (e) proceed to the left coronary plexus; a small number (o) passing to the superficial

¹ Quain's *Anatomy*, Ninth Edition, vol. i. p. 661, from which I have largely drawn in this description.

cardiac plexus. A few filaments (the small branch above the letter k in fig. 12), proceed to the anterior pulmonary plexus on each side.¹

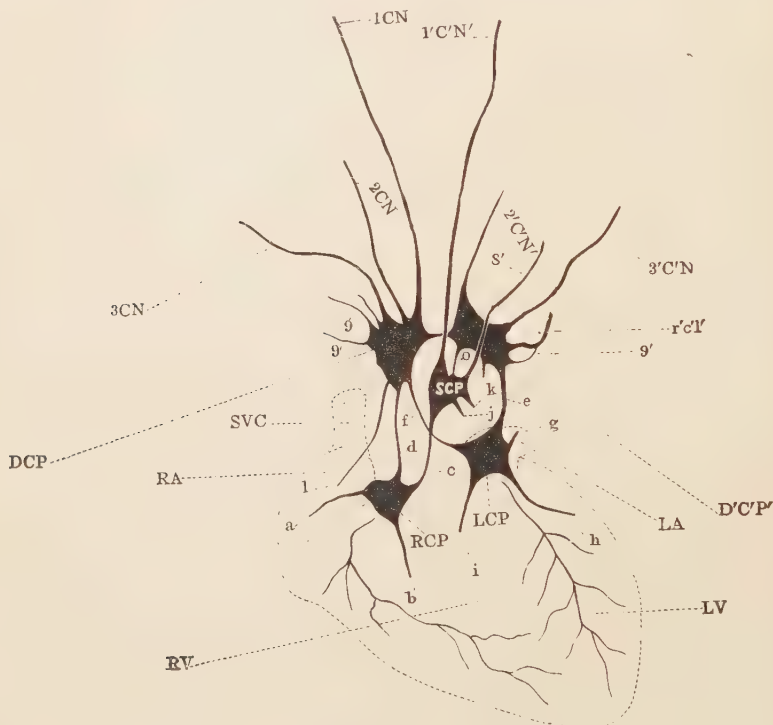


FIG. 10.—*Diagrammatic representation of the cardiac plexus. The different divisions of the plexus are shown as ganglion cells.*

SCP, superficial cardiac plexus; DCP, right, and D'C'P', left halves of the deep cardiac plexus; 1CN, first or superficial cardiac nerve on the right side; 1'CN', first or superficial cardiac nerve of the left side, 2CN, right, and 2'CN', left middle cardiac nerves; 3CN, right, and 3'CN', left lower cardiac nerves; 8', lower cervical branch of the left pneumogastric proceeding to the superficial cardiac plexus, 9, 9', branches of the right pneumogastric proceeding to the right half of the deep cardiac plexus; r'c'l' and 9', branches of the left recurrent laryngeal nerve proceeding to the left half of the deep cardiac plexus; c, branch from the superficial cardiac plexus to the right coronary plexus; j, k, branches to the pulmonary artery and pulmonary plexus of the left side. d and f, branches from the right half of the deep cardiac plexus to the right and left coronary plexuses, respectively; l, branch to the right auricle. e, branches from the left half of the deep cardiac plexus to the left coronary plexus; o, branch to the superficial cardiac plexus. The other letters have the same significance as in fig. 11.

¹ According to Pettigrew a few of the branches forming the superficial cardiac plexus pass backwards and appear on the posterior surface of the heart; while a certain number of the branches of the deep cardiac plexus pass forwards to appear on its anterior surface (*Physiology of the Circulation*, p. 298).

The cardiac plexus then is a 'junction' at which impulses passing to and from the heart may be transferred from one nerve path to another, and by means of which communications are established between the heart and distant parts. The 'main lines' which pass between the nerve centres and the heart are the sympathetic and pneumogastric. In order to complete the anatomical description of the cardiac nerves, I must now detail the origin and connections of their cardiac branches.

THE CONNECTIONS OF THE SYMPATHETIC WITH THE
HEART.

Each of the three cervical ganglia of the sympathetic sends a branch to the heart, the *upper*, *middle*, and *lower cardiac nerves* respectively (see fig. 11).

The upper or superficial cardiac nerve of the right side (I C N) arises by two or more branches from the upper cervical ganglion, and sometimes also by a branch from the trunk of the sympathetic, which joins the upper and middle cervical ganglia. After proceeding down the neck it enters the thorax, being directed along the innominate artery to the back of the arch of the aorta, where it terminates in the deep cardiac plexus (D C P). In its course through the neck and in the thorax, it forms numerous connections with other branches of the sympathetic and of the pneumogastric, the more important of which are—

1. A connection with the external laryngeal branch of the pneumogastric (1).
2. A connection with the trunk of the pneumogastric (2).
3. A connection with the recurrent laryngeal branch of the pneumogastric (3).

It also sends some small branches to the thyroid body, and to the front of the great vessels (aorta and pulmonary artery).

The upper or superficial cardiac nerve of the left side (1'C'N') arises in the same manner, and has the same course through the neck as its fellow of the right side. After entering the chest it is directed along the left common carotid artery to the front of the arch of the aorta, which it crosses, and terminates in the superficial cardiac plexus (S C P). In exceptional cases this nerve terminates in the deep cardiac plexus.

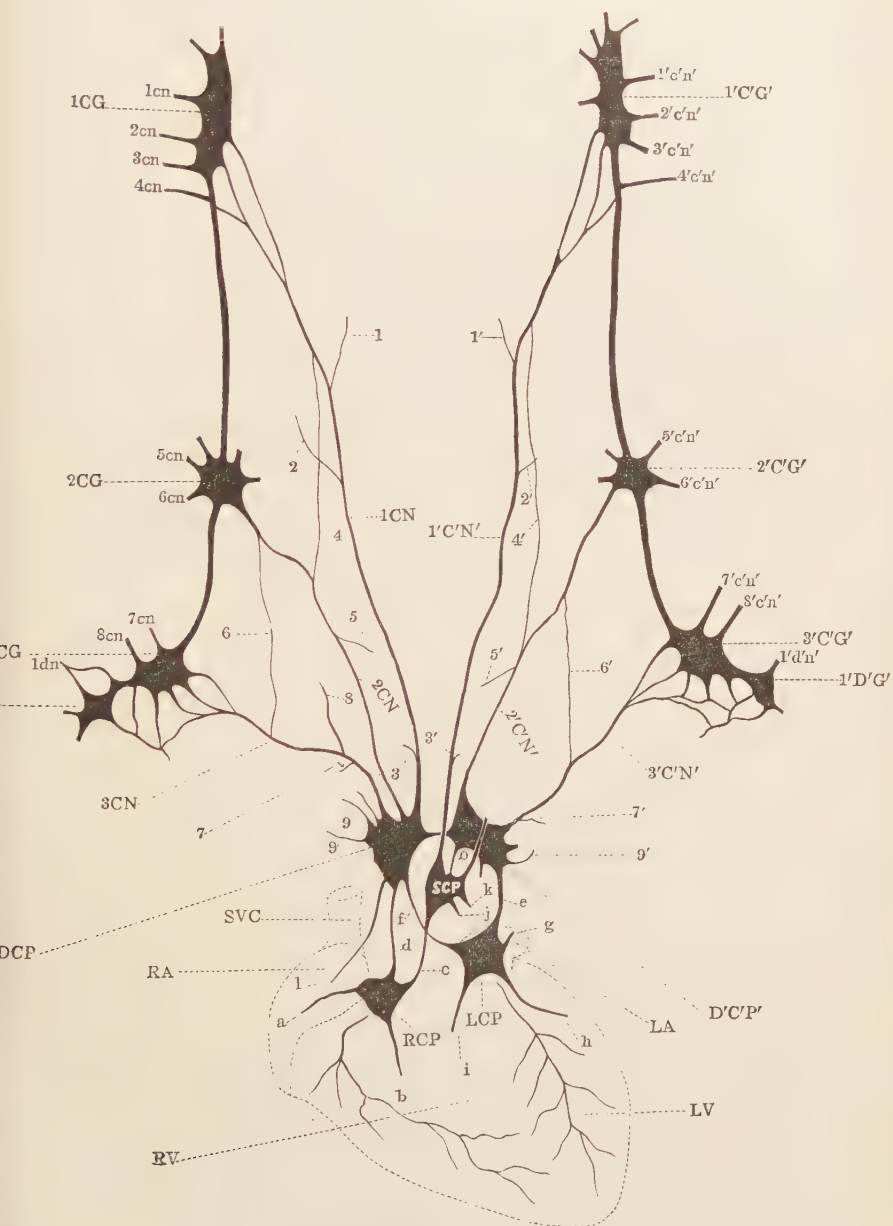


FIG. II.

THE CONNECTIONS OF THE PNEUMOGASTRIC WITH THE HEART.

Nerves destined for the heart arise from the trunk of the pneumogastric, both in the neck and in the thorax, and cardiac branches are also given off by several of its branches (see fig. 12). Anatomists consequently describe *cervical cardiac*, and *thoracic cardiac* branches of the pneumogastric.

The cervical cardiac branches.—In the upper part of the neck several small twigs (2) connect the pneumogastric with the upper cardiac branches of the sympathetic; while the external laryngeal branch of the superior laryngeal nerve, which is a branch of the pneumogastric, is also connected with the upper cardiac branch of the sympathetic (1).

In the lower part of the neck a branch of some size (8) arises from the trunk of the right pneumogastric, as it is about to enter the thorax. It is directed along the innominate artery to the back of the aorta, being usually blended into one of the branches of the sympathetic, and terminates in the right side of the deep cardiac plexus. This branch sometimes arises from the recurrent laryngeal.

The corresponding nerve of the left side (8') crosses in front of the arch of the aorta, and terminates in the superficial cardiac plexus.

Both nerves (right and left) give some small twigs to the coats of the aorta.

The thoracic cardiac branches.—On the *right* side several cardiac branches (9 and 9) arise from the trunk of the pneumogastric, and from the recurrent laryngeal, and pass to the right side of the deep cardiac plexus.

The corresponding branches from the left side (9') usually come entirely from the recurrent (Quain), and pass to the left side of the deep cardiac plexus.

In fig. 13 I have endeavoured to represent the nervous mechanism of the heart in all its complicated details.

Description of FIG. 12.—*Semi-diagrammatic representation of the connections of the pneumogastric with the heart.*

PP, right pneumogastric; P'P', left pneumogastric; rcl, rcl, right recurrent laryngeal nerve; r'c'l', r'c'l', left recurrent laryngeal nerve; sl, s'l', right and left, superior laryngeal nerves; el and e'l', right and left, external laryngeal nerves. 1 and 1', communicating branches between the external laryngeal nerves and the first cardiac nerves, on the right and left sides respectively; 2 and 2', between the trunk of the vagus and the first cardiac nerve on the right and left sides respectively; 8, communicating branch between the trunk of the right pneumogastric and the lower cardiac nerve; 8', on the right side this branch usually proceeds directly to the superficial cardiac plexus; 9, 9, branches from the right vagus, in the thorax, to the right half of the deep cardiac plexus; 9', branches from the left recurrent laryngeal to the left half of the deep cardiac plexus.

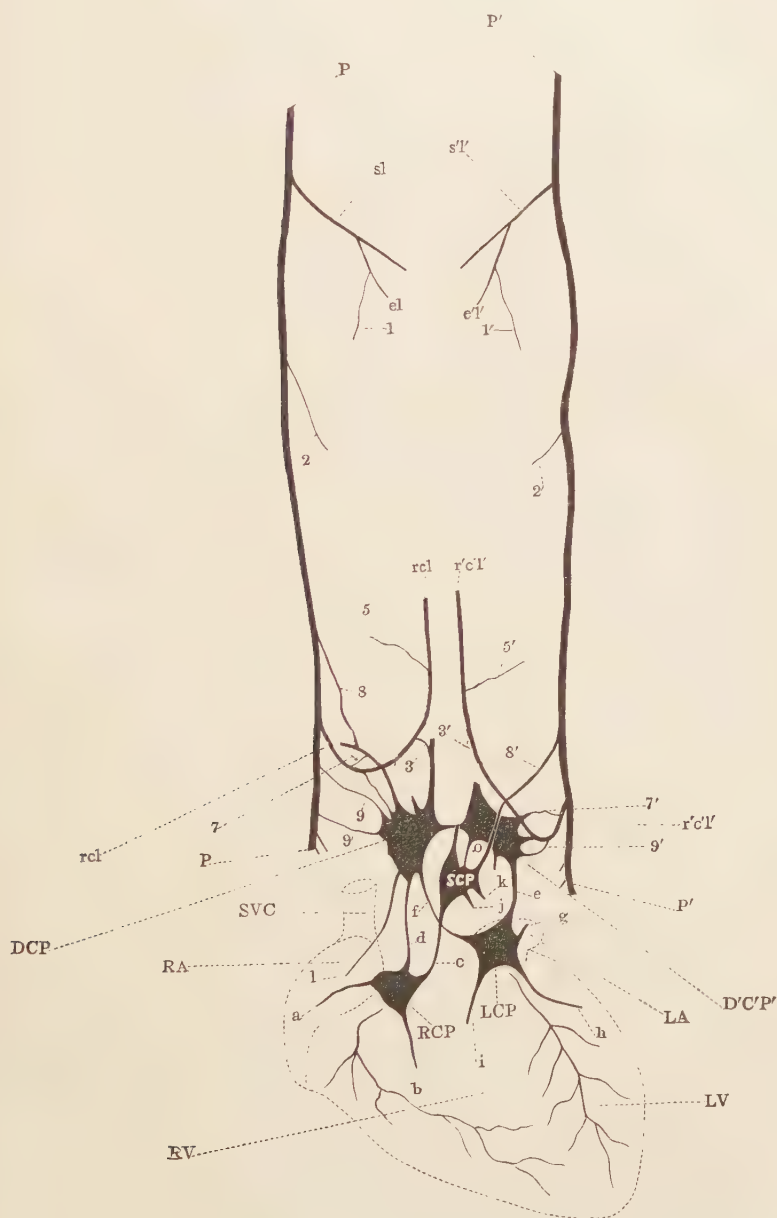


FIG. 12.

Description of FIG. 13.—Semi-diagrammatic representation of the nervous supply of the heart.

RCP, right, and LCP, left coronary plexuses; SCP, superficial cardiac plexuses; DCP, right, and D'C'P', left half of the deep cardiac plexus; P right, and P' left pneumogastric nerves; 1CG, Right, and 1'C'G', left superior ganglia of the sympathetic; 2CG, right, and 2'C'G', left middle ganglia of the sympathetic; 3CG, right, and 3'C'G', left inferior ganglia of the sympathetic; RV, right, and LV, left ventricles; RA, right, and LA, left auricles; SVC, superior vena cava.

1cn, 2cn, 3cn, 4cn, 5cn, 6cn, 7cn, 8cn, branches of the right, first, second, third, fourth, fifth, sixth, seventh, and eighth cervical nerves, proceeding to the sympathetic ganglia.

1'c'n', 2'c'n', 3'c'n', 4'c'n', 5'c'n', 6'c'n', 7'c'n', 8'c'n', branches of the left, first, second, third, fourth, fifth, sixth, seventh, and eighth left cervical nerves proceeding to the sympathetic ganglia.

1DG right, and 1'D'G' left first dorsal ganglia of the sympathetic. 1dn branches of the right, and 1'd'n' left first dorsal nerves proceeding to the first dorsal ganglia of the sympathetic.

sl right, and s'l' left superior laryngeal branches of the pneumogastric; EL right and E'L' left external laryngeal nerves; rcl right, and r'c'l' left recurrent laryngeal nerves at lower part of neck, and in the thorax respectively.

a, branch from right coronary plexus to right auricle; b, branch from right coronary plexus to right ventricle; c, branch from the superficial cardiac plexus to the right coronary plexus; d, branch from the right half of the deep cardiac plexus to the right coronary plexus; e, branch from the left half of the deep cardiac plexus to the left coronary plexus; f, branch from the right half of the deep cardiac plexus to the left coronary plexus; g, branch from the left coronary plexus to the left auricle; h, branch from the left coronary plexus to the left ventricle; i, branch from the left coronary plexus to the right ventricle.

1CN right, and 1'C'N' left, first or superficial cardiac nerves.

2CN right, and 2'C'N' left middle cardiac nerves.

3CN right, and 3'C'N' left inferior cardiac nerves.

1, branch from the right, and 1', branch from the left external laryngeal nerves to the superior cardiac nerves; 2, branch from the right, and 2', from the left pneumogastric nerves to the superior cardiac nerves; 3, communicating branch from the right, and 3', from the left recurrent laryngeal nerves to the first cardiac nerves on the right and left sides respectively; 4, communicating branch between the right, and 4', communicating branch between the left superior and middle cardiac nerves; 5, communicating branch between the right, and 5' between the left middle cardiac and recurrent laryngeal nerves respectively; 6, communicating branch between the right, and 6', communicating branch between the left, middle and inferior cardiac nerves; 7, communicating branch between the right, and 7', between the left inferior cardiac nerves and the recurrent laryngeal respectively; 8, communicating branch between the right inferior cardiac nerves and the trunk of the pneumogastric, 8', on the left side this branch of the pneumogastric proceeds directly to the superficial cardiac plexus; 9, 9, branches from the trunk of the right pneumogastric, and 9', from the left recurrent laryngeal nerve to the right and left halves respectively of the deep cardiac plexus.

THE MANNER IN WHICH THE VAGUS AND SYMPATHETIC
NERVES AFFECT THE HEART.

I have now described the construction of the mechanism concerned in the innervation of the heart, and I must next point out the manner in which that mechanism works.

We have already seen that while the movements of the heart are automatic (*i.e.* are due to impulses arising within the heart itself), they can be profoundly modified by the condition of distant parts; and that the impulses which produce these modifications are conveyed to the heart through certain branches of the pneumogastric and sympathetic nerves. Until recently, we might have summed up the effects of these impulses by saying, that *impressions passing to the heart through the pneumogastric, retard, while impressions passing to the heart through the sympathetic, accelerate its movements* (hence the terms '*inhibitor*' and '*accelerator*' of the heart which are given to the two nerves respectively); but recent observations seem to show that the action of the vagus on the heart is much more complicated, and that it affects not only the rhythm, but also the force of the cardiac contractions, and that it exerts a trophic influence upon the cardiac muscle. It will be necessary, therefore, to consider each of these functions separately.

The Inhibitory Action of the Vagus.

As the result of the laborious investigations of many able observers, physiologists have concluded:—*Firstly*, that there exists in the medulla oblongata a cardio-inhibitory centre, which is continually exerting a restraining influence upon the heart; and *Secondly*, that the action of this cardio-inhibitory centre may be intensified, *i.e.*, the action of the heart may be still further retarded by:—

(a) Direct stimulation, *i.e.*, by certain changes in the medulla itself.

(b) Impressions passing to it from the brain.

(c) Impressions passing to it from peripheral parts (reflex stimulation).¹

Some authorities also believe that the cardio-inhibitory centre may itself be inhibited, *i.e.* its restraining power taken off the heart, by impressions passing to the medulla from other parts of the central nervous system, or from the periphery.

The most important local changes which stimulate the cardio-inhibitory centre, appear to be increased blood-pressure within the cranium, and irritative lesions in the medulla or in the neighbourhood of the vagi roots, *e.g.* inflammatory affections at the base of the brain, etc.; while reflex inhibition of the heart seems to be chiefly brought about by impressions passing to the medulla from the abdominal viscera.²

It has also been supposed that the action of the heart may be inhibited by impressions passing from the heart itself. The experiments of Cyon and Ludwig, Rutherford and others, seem to show that reflex inhibition of the heart can be produced in the rabbit by stimulation of the central cut end of the superior cardiac nerve, the impression passing up to the cardio-inhibitory centre in the medulla oblongata, and back, through the vagus and its inferior cardiac branch, to the heart.

The *superior cardiac branch* arises by two roots, one connected with its superior laryngeal branch crosses down the neck close to the cervical sympathetic, and joins one or two branches of the inferior cervical ganglion with which it proceeds to the heart. This branch is a purely sensitive nerve; and is in fact the sensitive nerve of the heart. On

¹ Whether the action of the cardio-inhibitory centre is automatic or not, is still undecided.

² Powerful stimulation of any afferent or sensory nerve can probably produce reflex inhibition of the heart. Rutherford states that the cardio-inhibitory fibres of the vagus may be thrown into action by stimulating: (1). The central end of the vaso-inhibitory or superior cardiac branch of the vagus (depressor nerve); (2). The central end of the vagus of the opposite side; (3). Almost any sensory nerve, in the case of warm-blooded animals; (4). The abdominal viscera of the frog; (5). The splanchnic and sympathetic.—*Lancet*, Dec. 16, 1871, page 483. Michael Foster states that 'the regulative action of the inhibitory mechanism is brought into more or less close connection with all parts of the body.—*A Text Book of Physiology*, p. 174.

cutting it across, and stimulating its inferior (peripheral) end no effect is produced ; while stimulation of its upper (central) end causes pain, retardation of the heart, and, as I shall afterwards more particularly point out, such a striking diminution of blood-pressure, that the term *depressor* nerve has been given to it.

The *inferior cardiac branch* leaves the vagus below the origin of the inferior laryngeal nerve, and proceeds directly to the heart. It is a cardio-inhibitory nerve, and stimulation of its inferior cut-end gives rise to the same effects as stimulation of the vagus itself (*i.e.* stimulation above the origin of this branch), viz. (with weak currents) retardation of the heart from prolongation of the diastole, and (with stronger currents) complete arrest during diastole.

In the rabbit the vagus gives two branches to the heart, a superior and an inferior cardiac branch (see fig. 16).

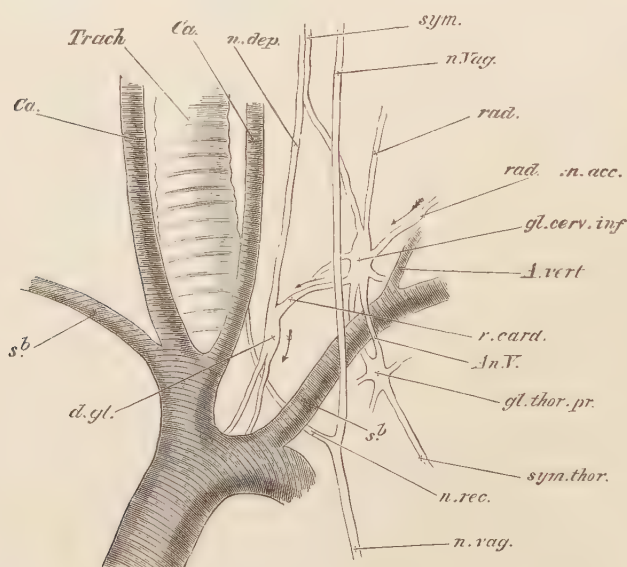


FIG. 14.—The last cervical and first thoracic ganglia in the rabbit. (Left side). (Somewhat diagrammatic, many of the various branches being omitted.)—(After Foster.)

Trach. Trachea. *Ca.* carotid artery. *s.b.* subclavian artery. *n. Vag.* the vagus trunk. *n. rec.* the recurrent laryngeal. *sym.* the cervical sympathetic nerve ending in the inferior cervical ganglion, *gl. cerv. inf.* Two roots of the ganglion are shown, *rad.*, the lower of the two accompanying the vertebral artery, *A. vert.*, being the one generally possessing accelerator properties. *gl. thor. pr.*

In the human subject, two branches of the vagus seem to correspond to the superior cardiac branch of the vagus in the rabbit; while the inferior cardiac branch of the vagus of the rabbit seems to be (mainly at all events) represented in man by the large branch which leaves the vagus trunk at the lower end of the neck (inferior cervical branch).

Now it has been supposed that these two nerves, together with the cardio-inhibitory centre in the medulla, constitute a reflex arc; and that stimulation of the peripheral terminations of the superior cardiac nerve in the heart may generate an impulse which, being conveyed to the cardio-inhibitory centre, and being reflected back to the heart along the vagus, may retard the action of the heart. Whether the superior cardiac nerve has any special function of this description, other than is possessed by all sensitive nerves, is extremely doubtful; and it seems certain that increased blood-pressure within the heart, as a whole, does not, as was at one time supposed, produce reflex inhibition of the organ.

Physiologists are not yet agreed as to the exact manner in which the vagus causes inhibition of the heart.

Until quite recently it was believed that while weak stimulation of the nerve prolonged the diastole, the duration of the ventricular systole being very little affected, more powerful stimulation caused complete arrest during diastole (complete relaxation), the next systole being as it were indefinitely postponed; and it was generally supposed that

Description of fig. 14 continued.

the first thoracic ganglion. Its two branches communicating with the cervical ganglion surround the subclavian artery forming the annulus of Vieussens. *sym. thor.* the thoracic sympathetic chain. *n. dep.* depressor nerve, which, though running by the side of the sympathetic, is really a branch of the vagus, from which it separates higher up. This is joined in its course by a branch from the lower cervical ganglion, there being a small ganglion at their junction, from which proceed nerves to form a plexus over the arch of the aorta. It is this branch from the lower cervical ganglion which possesses accelerator properties—hence the course of the accelerator fibres is indicated in the figure by the arrows.

the vagus inhibited the contractions of the heart by preventing motor impulses being sent from the automatic ganglia to the muscle, *i.e.* by holding in, as it were, the motor energy of the ganglia; and this would appear to be the view which Sewall and Donaldson take. They think that their experiments 'point pretty clearly to the conclusion, that the inhibitory action of the vagus nerve exerts itself in the same indirect way (as a motor stimulus does), influencing the ventricle by damping in sinus and auricles the excito-motor discharges arising in these parts of the heart.'¹

Dr Gaskell takes a different view. He has shown:—*Firstly*, that the vagus is able to modify all the great functional attributes of the cardiac muscle, *viz.*:—

(a.) The rate at which automatic contractions are produced. (*The automatic rhythm.*)

(b.) The force with which the contractions, more especially the contractions of the auricle, are carried out. (*The force of contraction.*)

(c.) The facility with which the contractions are conducted by the muscular fibres. (*The power of conduction*².)

Secondly, that it sometimes produces depression, at other times exaltation, of function.

He believes that the vagus may produce standstill or inhibition of the heart (auricles and ventricles) in the three following ways:—

(1.) By producing cessation of its automatic contractions, *i.e.* by depressing the rhythmical power of the muscular fibres of the sinus in which the automatic rhythm originates. (This has been established in the frog, tortoise, and snake.)

(2.) By reducing the force of the auricular contractions, so that they become invisible, *i.e.* by depressing the contraction power of the rapidly contracting reticulated muscular fibres of the auricle. (Frog.) The contractions, he states, may be

¹ *Journal of Physiology*, vol. iii. p. 367.

² In the tortoise the force of the contractions of the ventricle was uninfluenced by vagus stimulation. In the frog, on the other hand, the vagus influences the force of the ventricular as well as of the auricular contractions.

so small as only just to be visible, so that to the naked eye the heart appears to stand still.

(3.) By blocking the contraction wave at the sino-auricular ring, *i.e.* by depressing the conducting power of the muscular fibres connecting the sinus and auricle. (Snake, tortoise, frog.)

The Trophic Function of the Vagus.

As I have previously remarked, Dr Gaskell has advocated the important theory, which Eichorst had previously advanced, that the vagus is the trophic nerve of the heart. 'Although,' he says, 'the initial effect of the vagus is to depress some function, its final and most enduring power is to exalt, intensify, and repair that function. Thus, although it slows rhythm, yet its stimulation makes the rhythmical power last longer than it otherwise would, and makes the heart beat with regularity when it was previously irregular; although it reduces the force of the contractions, yet its ultimate effect is to improve and sustain the contraction force; although it may diminish the conduction power, yet in the end it completely repairs that power. *For these reasons,*' he says, '*I look upon the vagus as essentially the trophic nerve of the heart.*'¹

Since this chapter was written, I have learned from Dr Gaskell that the term 'trophic,' as it is commonly used and understood, does not exactly express the function which he supposes the vagus to have over the cardiac muscle. 'I have felt,' he says, 'all along that the word trophic is somewhat misleading as applied to the results of my experiments on the action of the vagus. I wanted a word to express, what appears to me to be the fact, in the cold-blooded animals at all events, that the inhibitory process is not destructive but constructive in its nature, but could not find any good term. I have therefore used the word 'trophic,' not perhaps in its usual sense, but rather in a sense similar to Heidenhain, when he divides the nerves supplying the sub-maxillary gland into secretory and trophic.'

¹ *Journal of Physiology*, vol. iv. p. 104.

The Mode of Action of the Vagus on the Heart.

The exact manner in which the terminal fibres of the vagus are brought into relation with the cardiac muscle is unknown. The effects produced by vagus stimulation, and by the action of certain poisons, have led some physiologists to believe that the arrangement is an extremely complicated one; and in order to explain these results, they have theorised that there existed an inhibitory mechanism within the heart itself, the action of which is strengthened (brought into play) by impressions carried to it by the terminal branches of the vagus, and which in its turn acts upon, *i.e.* inhibits the ganglionic apparatus more immediately concerned in the production of the rhythmical movements of the heart, *i.e.* the automatic nerve mechanism.

The facts and arguments which they have advanced in support of the view that the vagus does not act directly upon the cardiac muscle, but that it acts through some intermediate nerve apparatus, are as follows:—

1. The latent period, which elapses between electrical stimulation of the vagus and the production of its inhibitory action on the heart is twenty-times longer than that which elapses between the electrical stimulation of an ordinary motor nerve, and the contraction of its muscle, *viz.*, $\frac{1}{5}$ th and $\frac{1}{100}$ th of a second respectively.

2. A very much stronger current is required to produce inhibition of the heart through the vagus, than is required to produce spasm of a voluntary muscle, when its motor nerve is stimulated.

3. If the action of the heart be arrested by stimulation of the vagus, the rhythmical contractions after a time return, although the stimulation of the vagus be continually kept up, a fact which may be explained either by supposing that the inhibitory fibres of the vagus become gradually exhausted by the prolonged stimulation, or that the accumulation of nerve force in the automatic ganglia becomes after a time too great to be held back, and that an explosion and muscular contraction consequently take place.

4. Continued stimulation of one vagus annuls or prejudices the action of the other.

5. 'The effects of vagus inhibition are perhaps more marked when the electrodes are placed on the boundary line between the sinus venosus and the auricle, than over the vagus trunk itself.'—*Foster*.

6. If during the complete relaxation which results from powerful stimulation of the vagus, the muscular tissue be mechanically irritated, it is still found to contract.

While the existence of an intermediate (intra-cardiac) inhibitory mechanism between the terminal fibres of the vagus and the automatic nerve apparatus they think proved by the following circumstances :—

1. When the electrodes are placed on the boundary line between the sinus venosus and the auricles, more powerful inhibition is produced than when they are applied to the vagus itself.

2. After the administration of urari, stimulation of the vagus no longer produces inhibition of the heart, but stimulation in the region of the sinus venosus will still do so.

3. After the administration of atropia, neither stimulation of the vagus nor of the sinus produces any inhibitory effect. It is therefore concluded that atropia paralyses the intra-cardiac inhibitory apparatus itself.

4. Muscarin and jaborandi in full doses produce exactly the same effect which is produced by powerful vagus stimulation, viz., complete arrest during diastole, an effect which is not prevented by the previous administration of urari (which, as we have seen, paralyses the terminal fibres of the vagus, but does not touch the intra-cardiac inhibitory mechanism); but which is prevented by the previous administration of atropia which paralysis the intra-cardiac inhibitory mechanism. Hence it is concluded that muscarin and jaborandi stimulate the intra-cardiac inhibitory mechanism.

Dr Gaskell's view as to the mode of action of the vagus is different. He has shown that the action of the vagus on the cardiac muscle is in many respects identical with the action of a weak interrupted current. He believes that the nerve acts directly upon the muscular fibre itself, and he argues that the action of atropin, muscarin, and some of the other cardiac poisons to which I have referred above, can be consistently explained upon this view.¹

‘Again,’ he says, ‘I have shown clearly that the vagus depresses and exalts all the different functions of all the different muscular tissues of the heart, whether the function in question is rhythm, contraction, conduction, tone, or excitability. Also, I have shown that depression of one function is not necessarily accompanied by a simultaneous depression of another function, and so also with the exaltation of function. Further, the exaltation of each function is not necessarily dependent upon a previous depression; in each case the primary effect may, under certain circumstances, be

¹ *Journal of Physiology*, vol. iv., No. 2, pp. 114, 115, 116.

exaltation and not depression. Combining these two facts together, we are driven to accept one of two alternatives, either the vagus contains a multiplicity of fibres, which can be divided into two groups after the fashion of Heidenhain, 1. Depressors (*Hemmungs-fasern*); 2. Augmentors (*Verstärkungs-fasern*); and further, each member of each of these groups has again its own special function, so that, for instance, the rhythm-inhibiting nerve fibre is different in kind from the contraction-depressing fibre, and so on; or else, the same trophic nerve fibre produces all the different effects observed, according to the nature of the muscle, which it supplies, and the condition of that muscle at the time.

‘In this paper as well as in my previous paper, all the facts have tended strongly to prove that the vagus acts in the same manner upon the rhythm of the heart, and upon the force of its contractions, so that if separate inhibitory and accelerator nerve fibres exist for the one, separate depressor and augmentor fibres must exist for the other. If, therefore, it can be shown that such diminution and augmentation of the strength of the contractions are due not to different influences reaching the muscle, but to the same influence affecting the muscle when its conditions are different, then it is, to say the least, highly probable that slowing and acceleration are also due, not to different nerve fibres, but to the action of the same nerve fibre under different circumstances. In the frog, as I have previously pointed out, such a complete gradation exists between a primary excessive diminution of the contractions and a primary augmentation in consequence of nerve stimulation, as to render the hypothesis that such curves depend upon the simultaneous stimulation of two antagonistic nerve fibres very improbable. I have now proved in addition, that a weak interrupted current applied to the smallest strip of cardiac muscle produces the same two opposite effects, and that here the depressing effect may be removed and the augmenting alone remain, when the condition of the muscle is altered by the application of atropin. In order, therefore, to still hold to the view of specific nerve fibres acting in opposite directions upon the force of the contractions, it is necessary to

assume, not only that those nerve fibres possess opposite qualities up to their very termination in the muscle, but that even when the muscle itself is stimulated, the relative action of these two sets of nerves still holds its ground. In addition, atropin would have to be considered as acting upon these two sets of nerve endings, and not upon the muscle, paralysing the depressor nerve endings before those of the augmentor nerves. The experiments of Luchsinger and Szpilman,¹ which lead them to the conclusion that atropin has a special action upon unstriated muscle fibre, those of Bowditch, already referred to, and my own, all point to the conclusion that atropin affects the cardiac muscle directly in consequence of its affinity to unstriated muscle fibre. The whole evidence goes to show that the vagus is a constructive and not a destructive nerve, that the initial depression of function is not of the nature of exhaustion, but is preliminary to a greater functional activity. The phenomenon presented by the muscular tissue of the frog and tortoise under the influence of an induced current, is of exactly the same nature as that seen in the muscle of the snail's heart (as pointed out by Foster and Dew-Smith) when a constant or weak interrupted current is sent through it. In both cases augmentation of function occurs as well as depression; in the one case the evidence is as strong in favour of special inhibitory and accelerator nerves as in the other. Clearly, in the case of the snail, it is simply impossible, as Foster has said, to explain such results by the presence of accelerator and inhibitory fibres in every strip of muscular tissue, when, as a matter of fact, cardiac nerves of any kind whatever have not been proved to exist. Clearly also, whatever explanation will ultimately be found for the action of the current upon the muscular tissue of the snail's heart, will also explain the same phenomena in the heart of the frog and tortoise. The problem which demands solution is essentially, Why is the condition of the muscle ultimately improved in every one of its functions by the direct application to it of a continuous stimulus, which

¹ Pflüger's *Archiv.*, vol. xxvi. p. 459.

is not strong enough to produce motor effects? and why is that improvement of function preceded in many cases by a diminution of function? When these two questions have received a satisfactory answer, it will no longer be strange that the vagus produces throughout two opposing effects, and the action of atropin will become clear; then the relationship between trophic and motor action will be understood, and the true functions of the ganglion cells found in connection with nerve fibres will be indicated. At present it can only be said that the vagus is the trophic nerve of the cardiac muscle, its action resembling that of a stimulus too weak to produce motor effects, and therefore it is possible that the function of the ganglion cells, in the course of the nerve, is to convert an otherwise motor into a trophic nerve.¹ 'This argument,' Dr Gaskell writes me, 'is based upon experiments upon cold-blooded animals; how far it applies to the warm-blooded I (Dr Gaskell) do not yet know. Eichhorst and Zander conclude that the vagus in the warm-blooded contains trophic fibres.'

The action of the sympathetic upon the heart is even less clearly understood than the action of the pneumogastric, but it is believed that there exists in the medulla oblongata (or possibly above it), a centre, stimulation of which produces increased frequency of the heart's beat, and it is supposed that this accelerator, or cardiac-motor centre, as it is sometimes called, may be stimulated, *i.e.*, the frequency of the heart's beat may be increased either by direct or reflex stimuli. The influences which throw this centre into action have not been very accurately ascertained, but it is probable that emotion excites the heart through the agency of the accelerator nerves; while diminished blood pressure within the cranium is supposed to stimulate them.

Impulses generated in this centre, the action of which, be it observed, is supposed to be intermittent (and in this respect to differ from the action of the cardio-inhibitory centre, which is supposed to be constant), appear to pass down the lateral column of the cord to the lower end of the

¹ *Journal of Physiology*, vol. iv. p. 121.

cervical region, whence they pass through the nervi communicantes, to the (?) middle and inferior cervical ganglia of the sympathetic, and thence to the heart.

According to Prof. Michael Foster the accelerator nerves frequently pass along the nerves accompanying the vertebral artery, and reach the heart through the last cervical and first dorsal ganglia.¹

It is supposed from the long latent period which elapses between stimulation of the accelerator and the production of its specific action on the heart, that its fibres terminate in an intermediate ganglionic apparatus similar to that which I have already described in speaking of the termination of the pneumogastric.

Now, although the pneumogastric acts as inhibitor, and the sympathetic as accelerator, it must not be supposed that their action is directly antagonistic. That such is not the case seems abundantly proved by physiological observation. Physiologists have shown, says Dr M. Foster, that 'if during maximum stimulation of the accelerator nerves, the vagus be stimulated even with minimum currents, inhibition is produced with the same readiness as if the accelerator nerves were not acting. Vagus stimulation does not annul, but appears simply to suspend, during its continuance, the manifestation of the accelerator action.'²

It may appear to some that I have entered into undue physiological detail in dealing with this subject, but it must be remembered that it is only by attention to these minute physiological details that the physician can hope to make much advance in cardiac pathology, a subject on which our knowledge of the ordinary details of morbid anatomy is already so far advanced.

*The relationship between the Heart and the minute
Blood-vessels.*

In order to conclude the description of the innervation of the heart, I must shortly direct attention to the intimate relationship which exists between the heart on the one hand

¹ The middle and lower cervical ganglia in man appear to correspond to the lower cervical and first dorsal ganglia in the rabbit, through which accelerator impulses have been proved to pass, as shown in figure 14.

² *Text Book of Physiology*, Third edition, p. 175.

and the peripheral blood-vessels on the other, and to the beautiful self-adjusting mechanism by which alterations in the one are of necessity followed by corresponding changes in the other.

We have already seen the fundamental importance of looking at the heart both as a mechanical pump and as a vital organ; and another point which I must now insist upon—and it is hardly of less importance whether we are studying the heart from a physiological, a pathological, or a clinical point of view,—is *the necessity of taking an all-round view*, so to speak, *of the circulation*. Indeed it is essential to remember that the heart is only a part of the vascular mechanism, and that the condition of the circulation, and consequently the condition of the heart (for the organ naturally adapts itself to the amount and kind of work which it has to perform) depend in no small degree upon the condition of the peripheral resistance, which is in its turn mainly due to the obstruction which the blood meets with, in passing through the minute arteries.

In conditions of health, the minute arteries are always more or less constricted in consequence of a permanent contraction of their middle or muscular coats, and it is to this 'tonic' contraction that the peripheral resistance is in great part due. [The peripheral resistance depends also upon the facility with which the blood passes through the capillary system of vessels; and since the blood flow through the capillaries may be influenced by alterations in (a) the condition of the capillary walls, (b) the condition of the tissues outside the capillary walls, and (c) the composition of the blood itself, all of these factors must be taken into consideration as causes of peripheral resistance, and of variations in the blood pressure. In conditions of disease, variations in the blood pressure may result from alterations in the 'capillary resistance,' but these variations are, in my opinion, never so great, either in conditions of health or disease, as those which are due to alterations in the condition of the muscular coat of the minute arteries].

The degree of constriction is constantly undergoing variations even in health, and is subject to marked alterations in some diseased conditions; hence the amount of resistance which the heart has to overcome in forcing the blood into the capillaries, is constantly changing; and to meet sudden variations of this description a delicate self-adjusting nervous mechanism is provided. But in order that this part of our subject may be properly understood, I must now describe the mechanism by means of which the arterial 'tone' is regulated. There seems good reason to believe:—

(1) That the tonic constriction of the muscular coats of the minute arteries is (immediately) due to the presence of a peripheral neuro-muscular or (purely) muscular apparatus, which may be thrown into action by local stimuli, the most important of which is the blood pressure.

The peripheral mechanism concerned in this maintenance of arterial 'tone' probably bears a close resemblance to the peripheral mechanism concerned in the production of the rhythmical movements of the heart.

Ganglionic masses can be demonstrated in the walls of many of the minute blood-vessels, and it has been by some authorities supposed that the blood pressure produces stimulation of the muscular coats of these vessels in a reflex manner (see fig. 17); but in many vessels (*e.g.* those in the skin and muscle) no such ganglionic masses have as yet been demonstrated, and in their case we must conclude that the contraction of their muscular coats is due to direct stimulation of the muscular fibre itself, or of the terminal vaso-motor filaments which are distributed to it.

The view, which supposes that the muscular fibre is itself stimulated by the blood-pressure, is probably the correct one.

(2) That the action of this peripheral neuro-muscular or purely muscular mechanism is, under ordinary circumstances, maintained and regulated by impulses which are being *constantly* sent to it from centres (vaso-constrictor centres) in the medulla and spinal cord, through the vaso-motor nerves; and that the action of these vaso-constrictor centres may be increased or diminished both by direct stimulation or in a reflex manner. In other words, the calibre of the minute arteries, and therefore the peripheral resistance may be

increased or diminished by central and peripheral causes (stimuli).

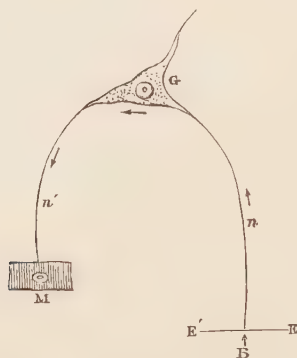


FIG. 15.—*Diagrammatic representation of the reflex mechanism, by which (it has been theorised) the muscular coat of the blood-vessels is thrown into contraction, under the influence of the blood-pressure.*

E' E, Endothelium; B, the blood in the blood-vessel; M, the muscular coat of the artery; g, ganglion cell—the reflex centre; n, sensory nerve fibre, conducting the impression generated by the blood pressure on the sensory nerve terminations in the wall of the vessel to the reflex centre; n', motor nerve fibril conducting the impulse from the reflex centre to the muscle.

The vaso-motor (*i.e.* vaso-constrictor) centre of the medulla, which is probably bilateral, is situated immediately above the calamus scriptorius, and extends for some distance into the pons (to 1 or 2 m m. below the corpora quadrigemina). The conducting fibres proceeding from it are believed to pass through the lateral columns of the spinal cord, and to proceed to the blood-vessels chiefly through the sympathetic system of nerves. The vaso-motor centre of the medulla probably regulates the vascular system of the abdomen, the arteries of the skin and muscles being probably mainly supplied by vaso-constrictor fibres proceeding from the vaso-motor centres in the spinal cord. The exact position of these spinal centres is not ascertained.

The action of the vaso-motor (*i.e.* vaso-constrictor) centres, which, it will be remembered, is supposed to be constant, may probably be intensified by :—

- (a) Increased blood pressure within the cranium, and local irritative lesions of the medulla.
- (b) Impressions proceeding from the cerebrum.
- (c) Impressions proceeding from the periphery. It has been experimentally proved that irritation of any afferent (sensory) nerve, such as the sciatic, will produce constriction of the minute blood vessels

(especially those of the abdomen) and increased blood pressure, presumably by reflexly stimulating the vaso-motor centre in the medulla.

(3) That the action of the peripheral neuro-muscular mechanism may be inhibited by nervous impulses passing to it from certain centres in the medulla, and (?) spinal cord, through the vaso-dilator nerves; and that the action of the vaso-dilator centres, which is not constant, may be called into play by central and reflex stimulation.

The calibre of the minute arteries may therefore be increased, and the blood-pressure lowered either by inhibition of the vaso-constrictor, or by stimulation of the vaso-dilator centres in the medulla and (?) spinal cord.¹

It is not absolutely certain that a distinct and separate vaso-dilator centre exists; some authorities in fact believe that the same centre may at one time evoke vaso-constrictor and at another vaso-dilator impulses; but it seems tolerably well ascertained that there are distinct fibres for the conduction of these two sets of impressions, and for theoretical and teaching purposes it is convenient to describe and represent a distinct vaso-dilator centre. The exact course of the vaso-dilator conductors in the cord is yet undetermined, but it seems probable that they pass down in some part of the lateral columns, and reach the blood-vessels chiefly through the cerebro-spinal (and not through the sympathetic) nerves. The vaso-dilator centre in the medulla is chiefly, if not exclusively, connected with the vessels of the abdominal viscera; and it is most readily thrown into action by stimuli passing to the medulla through the *depressor* nerve, and by impressions passing from the abdominal viscera through the splanchnics.²

¹ It must be remembered that the inhibitory action of the vaso-dilators on a peripheral neuro-muscular or muscular mechanism, is theoretical, and it is not definitely proved. 'It is quite possible,' as Foster says, 'that dilation may be brought about in different ways, in different cases, and so also with constriction.' — *A Text Book of Physiology*, p. 211.

² This description of the action of the vaso-motor mechanism is not complete. It must of course be remembered, that local variations in the blood supply are being constantly required by the ever varying needs of particular times and organs, and that as Prof. M. Foster says, 'the great use of the whole vaso-motor system is not to maintain a general arterial tone, but to modify according to the needs of the economy the condition of this or that vascular area' (*A Text Book of Physiology*, page 213). But in studying cardiac affections the changes in the action of the heart, which are induced by local variations of this description, may, for practical purposes, be safely ignored. Hence I have limited my description to those alterations of the vaso-motor mechanism which are sufficiently general to produce definite changes in the action of the heart.

Now, as I have previously stated, a very striking diminution of blood pressure results from stimulation of the superior cardiac branch of the vagus or *depressor nerve*, as it has therefore been called. It is obvious, therefore, that there is an important connection between the heart and the vaso-dilator centre in the medulla, by means of which the heart and peripheral blood vessels are brought into such close physiological connection, that changes at one end of the circulation, so to speak, are at once attended by compensatory changes at the other. If, for example, the arterial blood pressure is from any cause suddenly increased, a sudden strain is necessarily thrown upon the left ventricle, and it might easily happen that the resistance in front was too great for the force of the pump. Under such circumstances paralytic distention of the left ventricle and death would follow, if it were not for the fact that the undue distention of the cavity and the effort which the heart is making to overcome the resistance, produce irritation of the terminal fibres of its sensitive nerve (the superior cardiac or depressor nerve), in consequence of which a powerful stimulus is sent to the vaso dilator centre in the medulla, with the result that a sudden dilatation, of the capacious blood-vessels of the abdomen, occurs, and that there is a rapid fall of the blood pressure. The peripheral resistance is in this manner immediately diminished, and paralytic distention of the left ventricle prevented. [Where the arterial constriction and increased blood pressure are slowly and gradually established, compensation is effected in a different manner, viz., by the gradual increase in the strength of the pump, witness the hypertrophy of the left ventricle, which occurs in cirrhotic form in Bright's disease.]

It has been thought that the vaso-dilator and vaso-constrictor centres are also connected with the cardio-inhibiting centre in the medulla; and some physiologists have supposed that reflex inhibition of the heart might be produced by the stimulation of the sensitive nerve of the heart (superior cardiac nerve), which presumably occurs in conditions of over distention of the organ. The recent experiments, however, of

Ludwig and Luchsinger, and of Sewall and Donaldson, seem to prove that this is not the case, and that increased blood-pressure within the heart, as a whole, generally weakens the inhibitory influence of the vagus. The last two observers, however, conclude 'that changes of intra-cardiac pressure, *when experienced by the ventricle alone*' (no italics in the original), 'are without effect on the cardio-inhibitory function of the vagus.'¹ Possibly, therefore, when the ventricles only are over-distended, some reflex inhibition may occur; but, even if it does occur, it certainly must be temporary and slight, for the main effect of stimulation of the sensitive nerve of the heart is reflex stimulation of the vaso-dilator centre, and consequent diminution of blood-pressure throughout the body; and diminished blood-pressure, as we have previously seen, tends to lessen rather than to increase, the inhibitory action of the vagus.

So again, in some cases of sudden palpitation, the rise in blood pressure, which would necessarily be produced by the excessive action of the muscular pump, is possibly prevented by the stimulation of the cardio-inhibitory centre. *Vice versâ*, if from any cause the general blood pressure is suddenly diminished, the restraining influence of the cardio-inhibitory centre is removed, and the rapidity of the heart's action is increased—and the fall in blood pressure is to some extent at least counteracted.

¹ *Journal of Physiology*, vol. iii. p. 363.

CHAPTER II.

GENERAL PATHOLOGY OF THE HEART.

HAVING directed attention to some of the more important problems connected with the physiology of the heart, I will now briefly sketch the general pathology¹ of the organ. In considering the pathology of the heart, it is important to remember that it is a composite anatomical structure, and that it may be said to consist of three distinct parts, viz. :—

1. The pericardium.
2. The muscular substance or myocardium.
3. The endocardium.

The affections of the heart are either *functional* or *organic*; and I must now briefly describe the characteristic features of each of these great groups of conditions.²

The Functional Affections of the Heart.

The main characteristics of the purely functional³ affections of the heart are as follows :—

(1.) They have no distinct morbid anatomy; in other words, in the purely functional disorders no changes are to be found after death in the heart itself.

¹ The special pathology and morbid anatomy of the individual diseases which affect the heart, will be more conveniently considered when I come to treat of the individual affections in detail.

² *The distinction of organic and functional disease.* In cases of organic disease distinct structural changes are found after death, but in functional disorders no such changes can be demonstrated. It is impossible, however, to draw a sharp and absolute line of distinction between these two conditions, for every functional derangement does without doubt depend upon histological or chemical changes in the anatomical elements of the affected part, and it is probable that as our means of investigation become more minute, many of the so-called functional affections will be proved to be organic.

³ The purely functional affections, in which there is no distinct morbid anatomy, are practically synonymous with the neurotic affections of the heart.

(2.) They are often sudden in their onset, and usually temporary in their duration; they seldom if ever destroy life, and they are not, as a rule, followed by any permanent injurious effects.

Some of the so-called functional affections do occasionally result in organic disease; in exophthalmic goitre, for example, palpitation and accelerated action of the heart are prominent symptoms, and are for a time, at least, unattended by any perceptible physical alterations of the organ, but hypertrophy, and more especially dilatation, do in many cases ultimately occur.

(3.) They are, as a rule, due to derangement of the nervous mechanism of the heart; and the primary cause is very often located in some distant organ.

In some cases, the primary lesion (if we may use the term in connection with hysteria, hypochondriasis, etc.) is *cerebral*. Under this head are included the derangements of the heart which are so frequently met with in hysteria, hypochondriasis, and the like.

In others, the cervical portion of the *spinal cord* is the part at fault. Palpitation and extreme rapidity of the heart's action are occasionally met with, for example, in cases of myelitis and locomotor ataxy.

In a third group of cases, the primary lesion is situated in the cervical *sympathetic*; and in this group we are probably right in placing the derangements of the heart, which are such striking symptoms in the affection termed exophthalmic goitre.

In a fourth group, the derangement of the heart is due to *reflex* irritation, the primary cause being situated in some peripheral organ, such as the uterus or ovary.

While in other cases, the *intra-cardiac nervous mechanism* itself, or the muscular fibres, are directly affected; the palpitation, irregular action, etc., which result from the use of some drugs, should probably be included under this head.

(4.) The symptoms are usually referred to the heart itself, and consist of uneasy sensations, such as pain and palpitation. There are seldom any symptoms resulting from mechanical derangement of the circulation.

The Organic Diseases of the Heart.

The chief characteristics of the organic diseases of the heart are as follows :—

1. They have a distinct morbid anatomy.
2. They are both acute and chronic ; they are often permanent, or are usually followed by permanent structural defects ; they frequently shorten, and often destroy life.
3. They may affect either the pericardium, myocardium, or endocardium, in many cases all three structures being involved.

4. Their mode of origin is various, but the following main groups may be described :—

A. In some cases the disease is *primarily cardiac*. Under this head are included :—

(*a.*) The congenital malformations and imperfections, such as congenital stenosis of the pulmonary orifice and patency of the foramen ovale.

(*b.*) The mechanical ruptures of the valves which sometimes, though rarely, occur independently of any previous cardiac disease.¹

(*c.*) Primary new growths of the heart, such as primary cancer and primary sarcoma. These cases are extremely rare.

(*d.*) Primary (idiopathic) inflammations of the heart, *e.g.*, primary idiopathic pericarditis, primary idiopathic endocarditis, primary idiopathic myocarditis. These conditions are extremely rare, but they are occasionally met with. In many cases these so-called primary idiopathic inflammations are in reality rheumatic ; cases of pericarditis are, for example, occasionally met with, in which the cardiac inflammation is followed, instead of being preceded, by swelling of the joints and the other symptoms of acute rheumatism.

¹ In the large proportion of cases of rupture of the valves, which occur during violent effort, mental agitation, and the like, the valve, which gives way, is not absolutely healthy, but has been weakened by previous disease.

(*e.*) The local softenings, dilatations, and ruptures of the muscular substance which result from disease of the coronary arteries.¹

B. In others, the cardiac disease is part and parcel of a general affection.

Under this head are included :—

(*a.*) The inflammations of the pericardium, of the endocardium, and of the myocardium, which occur in the course of rheumatic fever, scarlet fever, Bright's disease, and many other general affections.

(*b.*) The softening and degeneration of the cardiac muscle which occur in cases of prolonged high temperature (pyrexia), notably in typhus.

(*c.*) The fatty degeneration of the heart, which is associated with general fatty changes throughout the body; and the fatty degeneration which is met with in conditions of anæmia, notably in the so-called progressive pernicious or idiopathic variety.

(*d.*) The waxy degeneration of the cardiac muscle, which is occasionally met with in the course of general waxy disease, but which is of little practical importance.

(*e.*) The ulcerative form of endocarditis, which is in some cases closely allied to pyæmia, and in which the cardiac affection, though undoubtedly the most prominent and central (or local) lesion, is, in many cases, only a local manifestation of a general septic condition.

(*f.*) The gummatous affections of the heart, which are occasionally seen in tertiary syphilis.

(*g.*) The hypertrophy of the left ventricle, that results from obstructed arterial circulation, and which is seen in its most typical form in the cirrhotic form of chronic Bright's disease; and the dilatation and hypertrophy of the right ventricle, which result from obstructed pulmonary circulation,

¹ Although I have placed these cases under the primary affections of the heart, it must be remembered that the disease of the coronary arteries, which gives rise to them, is usually part and parcel of a general arterial affection (*e.g.* atheroma). From this point of view, therefore, we might include local softenings, etc., in the second great group of cases.

notably in connection with emphysema, cirrhosis of the lung, and the pulmonary congestion produced by mitral lesions.

Many of the chronic affections of the heart, which at first sight appear to be primarily cardiac, *i.e.* to originate in the heart itself, should be included in this group. Chronic valvular lesions, for example, sometimes owe their origin to a previous attack of rheumatic fever and endocarditis. Fibroid degeneration, too, often results from a previous myocarditis; while the rare condition, aneurism of the heart, is usually produced by a local fibroid change, which in its turn may have resulted from a previous myocarditis. In other cases, valvular lesions (aortic valvular lesions more particularly) are due to atheromatous changes at the base of the aorta, which are part and parcel of a general arterial disease.

C. In a third group of cases the disease of the heart is due to extension to the heart of a morbid process which has its original seat in some other organ. The extension may take place—

(*a.*) By direct continuity of tissue, as, for example, in those cases in which a pericarditis results from an inflammation of the pleura, or in which a mediastinal growth, a lympho-sarcoma, for instance, makes its way through the pericardium and involves the heart.

(*b.*) Indirectly through the veins or lymphatics, as in those cases in which secondary tubercles, abscesses, cancers, or hydatids form in the substance of the heart or in the pericardium, the infective particles being carried to the heart from the lung or some other more distant organ.

5. The most prominent symptoms (dropsy, cough, shortness of breath, etc.) are generally due to derangement of the venous or arterial circulation of distant parts or organs; the symptoms, referred to the heart itself, being in most cases comparatively insignificant.

Now, organic lesions of the heart chiefly affect the circulation in two ways, viz. :—

A. *By impairing the force of the cardiac muscle (cardiac pump).*

Lesions, such as fatty, fibroid degeneration, etc., which weaken the cardiac muscle must of course impair its 'driving' power, and of necessity produce retardation of the circulation. Lesions of this description usually involve both sides of the heart (both the right and left hearts), and affect more particularly the muscular walls of the ventricles. When the strength of the left ventricle is impaired, the amount of blood pumped into the arterial system is less than normal, while the left auricle, the lungs, the right heart, and the systemic venous circulation become over-distended, and the rapidity of the whole circulation is decreased. When again the wall of the right ventricle is weakened, the amount of blood supplied to the lungs, left side of the heart and systemic arterial circulation is deficient, while the systemic venous circulation is over-distended.

It is important, too, to remember that when the cardiac walls are weakened, the venous ostia (mitral and tricuspid orifices) are less firmly closed than in health, and a leakage through these orifices is apt to take place. This is probably the chief cause of the mitral regurgitation which occurs in chlorosis and many other conditions; and this form of incompetence we may conveniently term '*muscular*,' *i.e.* incompetence due to defective muscular closure of the valvular orifice.

Then again, when the cardiac walls are weakened, the blood pressure, which in conditions of health is opposed by the elastic resistance of the cardiac muscle, readily produces dilatation of the cardiac cavities, and this condition, *i.e.* dilatation, materially adds to the difficulties of the circulation, for the following reasons:—

(a.) In proportion as the dilatation increases, the walls of the cavity become thinner, and therefore weaker.

(b.) The greater the amount of blood which the cavity contains, the greater the amount of force required to empty it.

(c.) Dilatation of the ventricles, by preventing the perfect

closure of the auricula-ventricular valves permits of regurgitation, and to this condition the term '*relative incompetence*' is usually applied. It is probable, however, that in many cases of dilatation, defective muscular closure (the condition which produces '*muscular incompetence*,' as I term it) is a more important cause of regurgitation than the actual stretching of the valvular orifice itself.

In dilatation, then, the muscular wall is not only weaker than in health, but a greater amount of work is actually demanded of it; and the circulation may be still further embarrassed by regurgitation through the auriculo-ventricular orifices.

It is extremely important to remember that the regurgitation which results from 'defective muscular closure,' and from 'relative incompetence,' is not necessarily a permanent condition. Indeed in many cases we can, by appropriate treatment, restore the cardiac muscle to its previously healthy state, and with the restoration of the cardiac muscle the regurgitation necessarily disappears.

The condition, therefore, of the cardiac muscle, and the presence or absence of dilatation, are points of the greatest practical importance. Indeed, as we shall afterwards see, the prognosis is largely based upon the condition of the heart in these respects; and the treatment of cardiac affections is, to a great extent, directed to maintaining the cardiac muscle in a sound and healthy state.

B. By producing structural alterations in the valvular orifices, and valve flaps, which interfere with the valvular mechanism, and prevent the steady onward flow of blood in the natural course of the circulation.

These alterations constitute the great group of valvular lesions properly so called. They are usually permanent and incurable. They consist of adhesions, thickenings, contractions, and ulcerations of the valve-flaps and adjacent parts, in consequence of which, narrowing (*stenosis*) of the valvular orifice, or imperfect closure of the valvular apparatus (*incompetence*) is produced. The two conditions (*stenosis* and *incompetence*) are very generally combined; in some cases *stenosis*, in others *incompetence* being the more prominent.

Both conditions (stenosis and incompetence) interfere with the steady onward flow of blood in the normal direction, and produce diminished supply of blood to the parts in front of the lesion and vascular engorgement of the parts behind. But the manner in which these effects are produced differs somewhat in the two cases. In stenosis the obstruction is *passive*, the blood simply stagnating, as it were, in the cavities of the heart and parts of the circulation behind the 'block;' while in incompetence the obstruction may be termed *active*, for it is due to a 'backwash' or regurgitant current, which presses back, as it were, and arrests the advance of the blood column into the cavity of the heart, which is situated immediately behind the seat of the lesion.

But further, the regurgitant current, passing as it does with considerable force into a cavity, the walls of which are relaxed and flaccid, has a stronger tendency to produce dilatation of that cavity than simple passive obstruction has—witness the condition of the left ventricle in aortic stenosis and incompetence respectively; and dilatation adds, as we have already seen, very materially to the difficulties of the circulation.

Now, from what I have said, it might be supposed that every structural alteration which produces either stenosis or incompetence of a valvular orifice, is necessarily attended by symptoms due to disturbance of the circulation; and such in truth would be the case, if it were not for the fact, that nature adapts herself to the altered condition of things; and that certain secondary changes are gradually established, by virtue of which the bad effects of derangement of the circulation are resisted, and by means of which the normal balance, so to speak, of the circulation is maintained or re-established. There is, in short, in almost all cases of valvular defect, a natural effort to *compensate the lesion*, the importance of which in a practical point of view, it is impossible to over-estimate.

These compensatory changes consist of alterations in the heart, the object of which is to restore and maintain the balance of the circulation, and to resist the injurious effects of the lesion on the heart itself; and of certain changes in

the peripheral tissues, by means of which the injurious effects of backward pressure and venous stagnation are, to some extent, prevented.

The exact nature of these compensatory changes, which depend upon (1.) the valve which is affected; and (2.) the manner in which it is affected (*i.e.* whether stenosis or incompetence, is the chief lesion), will be more appropriately considered when I come to treat of the individual valvular lesions in detail; but, speaking broadly, I may say that in all valvular lesions compensation is *chiefly* effected by hypertrophy of the walls of the cardiac cavity or cavities, which are situated behind the affected orifice.¹

When, for instance, the aortic orifice is contracted, the muscular wall of the left ventricle becomes thicker, and the 'driving' power of the left heart being materially increased, a larger quantity of blood is propelled in a given time through the narrowed orifice than could possibly have been the case in the normal (un-hypertrophied) condition. So again stenosis of the mitral valve is followed by hypertrophy of the left auricle, but in this case (the normal function of the auricle being passive rather than active, and the resisting power of its walls—against the blood pressure—depending not only upon the muscular tissue but also upon the connective tissue layers of the endocardium), the hypertrophy consists not only of an increase of the muscular wall of the auricle, but also of thickening of its elastic tissue lining. By these means its resisting power is materially strengthened at the same time as its propelling power is increased.² This increase of the connective tissue coat of the auricle is (in proportion to the amount of muscular hypertrophy) still more marked in mitral

¹ Alterations in the frequency of the cardiac contractions also exert an important compensatory influence, more especially, as we shall afterwards see, in the case of aortic lesions.

² The reader must not suppose from this statement that all fibroid changes *in* the cardiac walls add to the resisting power of the organ. When the muscular tissue of the organ is replaced by fibrous tissue, as it is in fibroid degeneration, both the 'driving' and resisting power of the organ are diminished. It is only when the muscular wall remains healthy, or is hypertrophied, that an increase of the fibrous tissue in the endocardium can possibly add to its resisting power.

incompetence, in which condition, as we have previously seen, increased resistance is necessary to counteract the dilating force of the regurgitant current, but in which there is no obstruction to the passage of the blood from the auricle to the ventricle. So again in aortic regurgitation, the forcible passage of an abnormally large quantity of blood into the cavity of the left ventricle during its diastole (from the aorta through the incompetent valve, and from the left auricle through the mitral orifice), produces over-stimulation of the muscular fibre, in consequence of which, hypertrophy of the left ventricle is produced; and this for a time, at least, is able to counterbalance the bad effects of dilatation, a condition which is produced, as we have already seen, by the too forcible distention of the cavity while its walls are flaccid and relaxed.

The hypertrophy, then, which follows and accompanies valvular lesions, is eminently beneficial, though it is not in all cases an unmixed good; and I cannot insist too strongly upon the immense importance of this doctrine of compensation. The symptoms, as we shall afterwards see, are trivial, or altogether absent, so long as the compensatory changes are sufficient to balance the bad effects of the lesion; the prognosis is very largely based upon the amount of compensation and the capabilities of repair which are present; while the treatment is in great part directed to promoting and maintaining the hypertrophy and other secondary changes, by means of which the balance of the circulation is restored and maintained in a comparatively normal condition.

The amount of compensation which is possible in any given case, depends chiefly upon the following circumstances:

1. *The suddenness, extent, and character of the lesion.*

A very extensive lesion, which occurs suddenly—rupture of the heart, for instance—may of course destroy life so rapidly that compensatory changes cannot possibly occur.

Then again a severe (but not immediately fatal) lesion which occurs suddenly, is with difficulty compensated—ruptures and ulcerations of valves are examples.

In other cases, on the contrary, in which the progress of the lesion is slow and gradual, compensation is easily

established, and is very complete. In many chronic valvular lesions, for example, compensatory changes advance *pari passu* with the morbid process, and for a time at least, the balance of the circulation is so satisfactorily maintained, that the patient (provided that he lives a quiet and tranquil life, and does not suddenly add to the difficulties of the circulation) may be unaware of the existence of any cardiac defect.

2. *The reparative powers of the patient, and especially the capabilities of compensation existing in the heart itself.*

3. *The resisting power of the tissues*, which in its turn depends upon the soundness and vitality of the individual organs, and especially upon the vaso-motor nerve tone, and the vitality of the whole organism.

In young persons, where the tissues are healthy, and in persons of good nerve tone and tranquil disposition, compensation is satisfactorily, and, for a time at least, effectually established. *Vice versâ*, in old people, in persons whose tissues are unsound or degenerating, more especially in those in whom the nerve tone is bad, compensation is, from the first, imperfect, and the injurious effects of the lesion are speedily manifested in the form of symptoms.

The condition of the tissues, then, as a whole, and the reparative power and vitality of the patient are facts which the practical physician must ever keep prominently in view. Indeed, we may lay it down as an axiom, that *in looking at cardiac cases, whether from a pathological or a clinical point of view, and more especially in considering the prognosis and treatment, it is quite as important* (I might even say that in some cases it is more important) *to look at the condition of the system as a whole, as it is to regard the condition of the heart in particular.* He is in fact a poor physician who concentrates his attention upon the tissue or organ which is primarily affected; and this statement holds good even should he succeed in arriving at an accurate estimate of the cardiac or other local lesion; while the best physician is he who accurately gauges the nature and extent of the local lesion, and at the same time takes a broad and comprehensive all-round view of the case.

CHAPTER III.

THE CLINICAL INVESTIGATION OF CASES OF CARDIAC DISEASE—
METHOD OF CASE-TAKING—SUMMARY OF SYMPTOMS—THE
PHYSICAL EXAMINATION—INSPECTION—PALPATION—PERCUSSION—
AUSCULTATION—THE USE OF THE SPHYGMOGRAPH.

HAVING considered some of the most important points connected with the physiology and general pathology of the heart, we are now in a position to take up the clinical examination of the organ. And in order to make this most important part of our subject as clear and intelligible as possible, I will first sketch out the method of case-taking, and the plan of examination which I am in the habit of employing in the investigation of cases of cardiac disease—and we shall, then, be in a position to consider the symptomatology and physical examination in detail.

Under the head of the physical examination I shall:—

1. Describe the normal physical signs (*i.e.* the signs appreciable to the senses—aided and unaided—of the physician, which result from the physical condition of the organ in its healthy state), and their mode of production.

2. Describe the pathological physical signs (*i.e.* the signs which result from the physical condition of the organ when diseased) and their mode of production.

3. Give in a short and summary manner, the leading facts, which will enable us, in any case in which an abnormal physical sign is detected, to determine the nature of the lesion which is present; for it is important to remember that few physical signs are absolutely distinctive (pathognomonic); in fact, diseased physical signs, such as increased dulness on percussion over the præcordia, may be due to several different morbid conditions.

METHOD OF CASE-TAKING.

A. PRELIMINARY FACTS :—Name—Age—Sex—Married or Single—Occupation—Full Postal Address—Date of Admission to Hospital.

B. COMPLAINTS :—(The Symptoms which bring the patient to consult the physician).

C. THE HISTORY :—

(1.) **Of the Present Illness :—**The exact date of its commencement. The exact mode of commencement. The supposed cause of the attack. The exact character of the symptoms; the order of their appearance; and the treatment which has been adopted, up to the time when the patient comes under observation. (In acute cases take the temperature.)

(2.) **The Health History prior to the commencement of the present attack :—**Especially a history of disease or injury likely to be followed by disease of the heart. The habits, mode of life, and general surroundings of the patient.

(3.) **The Family History :—**Especially the occurrence of heart affections or of acute rheumatism amongst near relatives.

D. THE PRESENT CONDITION :—(The date at which the examination is made should be stated.)

(1.) **The Physiognomy of the Case :—**The description of any striking abnormal appearances. The condition of the superficial vessels. The character of the breathing. The presence of subcutaneous oedema. The attitude. The general state of nutrition. The facial expression, etc.

(2.) **The presence or absence of subjective symptoms referred to :—**

(a.) *The Heart itself.*—(Uneasy sensations in the region of the heart, palpitation, pain, etc.)

(b.) *Distant Organs or Parts.*—(Symptoms resulting from mechanical disturbance of the circulation—deficient supply of arterial blood, or venous engorgement, etc.)

(3.) **The Physical Examination of the Heart and Circulatory Organs.**

The Physical Examination of the Heart.*Inspection of the Præcordial Region.*

Observe :—

(a.) Its form and configuration.

(b.) The position, extent, and character of the visible impulse, especially the position of the apex beat.

(c.) The condition of the integument over the præcordial region.

Palpation of the Heart.

Note :—

- (a.) The exact position of the apex beat.
- (b.) The character of the cardiac contractions (force, rhythm, celerity, etc.)
- (c.) The presence of præcordial thrills or friction fremitus.
- (d.) The presence of pain or tenderness on pressure over the præcordia.

Percussion of the Heart.

Determine :—

- (a.) The area of superficial or absolute dulness.
- (b.) The area of deep or relative cardiac dulness.

Auscultation of the Heart.

Determine :—

- (a.) The rhythm of the heart, whether regular or not.
- (b.) The character of the individual (first and second) sounds in the mitral, aortic, pulmonary, and tricuspid areas, as regards :—
 - Loudness or intensity.
 - Tone and purity.
 - Reduplications.
 - Murmurs.
- (c.) Where a murmur is present, observe :—
 - Its rhythm.
 - Its point of differential maximum intensity.
 - The direction in which it is propagated.
 - Its sound characters.

The Physical Examination of the Aorta and Great Vessels.

Inspection.

The conformation of those parts of the thorax and abdomen which are superficial to the aorta and great vessels, must be observed, particularly the presence of any prominence, pulsation, or tumour.

Palpation.

Determine :—

- (a) The presence of any undue pulsation in the supra-sternal notch, or of any abnormal pulsation in the thorax or abdomen.
- (b) The presence of thrills, tenderness on pressure, etc., in the course of the aorta or great vessels.

Percussion.

Note :—

The presence of dulness on percussion in the course of the aorta or great vessels; its exact extent, outline, etc.

Auscultation.

Observe :—

The character of the heart sounds, and the presence or absence of murmurs over the course of the aorta or great vessels (their rhythm, direction of propagation, etc.)

The Examination of the Superficial Arteries.

Note the condition of the superficial arteries, such as the carotids, brachials, temporals, etc., by *inspection*, and if necessary by *palpation* and *auscultation*; and particularly observe the condition of *the* pulse (*i.e.* the radial artery):—

(*a.*) By the finger (palpation) as regards:—

Its frequency.

Its rhythm.

Its volume.

Its compressibility or tension.

The special characters of each pulse wave (celerity, dirotism, etc.);

and the condition of the vessel (in respect to its fulness) during the diastole of the ventricle.

The condition of the arterial coats.

The comparative conditions on the two sides of the body (*i.e.* the comparison of the two radial arteries).

(*b.*) By the eye (inspection).

(*c.*) By the sphygmograph.

The Examination of the Venous System.*Inspection.*

Note the condition of the superficial veins, and particularly observe the condition of the jugulars as regards fulness, the presence of pulsation, etc.

Auscultation.

Note the presence or absence of a venous hum in the neck, over the orbit, Torcular Herophili, etc.

(4.) The condition of the Respiratory, Alimentary, Genito-Urinary, Nervous, and Integumentary Systems.

E. THE DIAGNOSIS.**F. THE PROGNOSIS.**

G. THE TREATMENT—Hygienic, Dietetic, Medicinal (general and local).

H. THE SUBSEQUENT COURSE OF EVENTS.

The progress of the case during the patient's stay in hospital. The mode of termination of the case. The date of termination. In fatal cases the record of the *post-mortem* examination, and an account (when necessary) of the subsequent microscopical examination of the tissues and organs.

SUMMARY OF THE CHIEF SYMPTOMS.

AGE.—Diseases of the heart may occur at any age, but some affections are more common at one period of life than at another.

Childhood.—*Congenital Malformations* are generally attended with symptoms, and are, therefore, as a rule, observed immediately or soon after birth. Many cases prove fatal during the first few weeks or months of extra-uterine existence; and severe cases, which do not die at an early stage, usually succumb at or about the time of puberty. Congenital malformations are, therefore, rarely noticed, except on the *post-mortem* table, in the adult.¹ *Acute inflammations*, more especially endocarditis, are sometimes seen in children, and are probably of much more frequent occurrence than is generally supposed; and cases of valvular disease are by no means rare before the age of puberty; mitral lesions frequently follow scarlet fever, and mitral regurgitation is present in a considerable proportion of the cases of chorea.

Youth and Early Adult Life.—*Functional* affections of the heart are most common from the age of puberty up to the age of twenty-five or thirty, but are also, of course, met with in later life. *Acute inflammations* of the heart, more especially those forms which are associated with rheumatism, are also most common in early adult life. The valvular lesions, more especially the lesions of the mitral valve, which so often follow acute endocarditis, are consequently of frequent occurrence at this time.

Active manhood.—The lesions of the heart and arteries, which are due to strain, syphilis, and drink, now begin to appear, aortic valvular lesions and aneurisms being especially prominent. The forms of disease, which are prevalent in early adult life, also occur.

¹ Congenital lesions of the valves which have not interfered with the circulation, and which were not suspected during life, are not unfrequently discovered after death. In cases of this description the congenitally malformed valve is frequently the seat of disease acquired in later life—of endocarditis, chronic valvular lesions, etc.

Later Adult Life and Old Age.—Valvular lesions, which in some instances date from a former attack of acute rheumatism, and have been long latent, now begin to be actively manifested; and all the other lesions which depend upon degenerative changes either in the heart or arteries become prevalent. *True angina pectoris* is seldom observed before the age of forty; *fatty degeneration of the heart, dilatation, and valvular lesions* of all kinds, are extremely common, and cause a large proportion of the deaths between the ages of fifty-five and sixty. In women the period between forty-five and fifty, and in males between fifty-five and sixty, seems a particularly critical one. The tendency to aortic valvular lesions, aneurism, and all the other cardiac changes which depend upon atheroma, steadily increases with the age of the individual; and these affections (aneurism, apoplexy, etc.) would be still more prevalent if it were not for the inactive (strainless) lives which most old people lead, the tendency to arterial rupture being in consequence reduced (considering the condition of the arteries) to a minimum.

SEX.—The *functional* derangements of the heart, which accompany *hysteria* and *anæmia*, and the cardiac affections which are met with in *chorea* and *exophthalmic goitre* are very much more common in the female sex.¹ Those cardiac affections, on the contrary, which are due to atheroma and gout, to strain, syphilis, and alcohol, are very much more prevalent in males; aortic lesions, aneurisms, and true angina pectoris are examples in point.

OCCUPATION.—The occupation of the patient exercises a very distinct influence in the production of cardiac disease. Persons who are exposed to cold and wet, such as washerwomen, firemen, cabmen, etc., are much more liable to contract rheumatic fever, and therefore to suffer from acute inflammatory affections of the heart and the chronic valvular

¹ Hysteria, chlorosis, chorea, and exophthalmic goitre are all much more common in females.

lesions, which so frequently supervene upon acute endocarditis, than other people. Blacksmiths, puddlers, paviors, and all persons engaged in laborious occupations, more especially those who have to make sudden efforts, and who are therefore exposed to strain, are more liable to atheroma, aortic valvular lesions, and aneurism, than other people. Soldiers and sailors are also very often affected with arterial disease probably because they are much exposed to strain, and because they are more subject to syphilis than other members of the community, and are also frequently addicted to alcoholic excesses. The constriction of the neck and chest, which is produced by tight or badly fitting clothing and accoutrements, is probably also another cause for the prevalence of aneurisms amongst soldiers; the constriction produces a direct mechanical impediment to the circulation, which is increased by any strain or violent exertion, such as a charge at 'the double,' or a 'forced march.'

COMPLAINTS.—In order to understand the 'complaints' of the patient, it is essential to remember that the heart is a vital organ as well as a mechanical pump. There are, in short, two great groups of cardiac symptoms.

(1) In one, the symptoms are referred to the heart itself, and depend upon the fact that the patient is conscious of some derangement in its action as a vital organ. These symptoms, which may for the sake of convenience and description be termed '*vital;*' or better, '*subjective cardiac sensations,*' consist of:—uneasy sensations in the region of the heart; consciousness of excessive or disordered action (palpitation, irregularity, intermittent action, and the like); and in some cases, severe pain in the cardiac region.

(2) In the other group, the symptoms are referred to some distant organ or part, and depend upon the mechanical derangements in the circulation which are produced by the cardiac lesion (*i.e.* derangement of its action as a mechanical pump). Giddiness, fainting, cough, shortness of breath, dropsy, dyspepsia, drowsiness, etc., are examples of this group, to which the term '*mechanical*' may be applied.

The mechanical symptoms are, for the most part, due to one or other of two conditions, viz. :—

(a) *Defective supply of arterial blood to distant organs and parts.*—The attacks of giddiness, fainting, the feeble motor power in the lower extremities, and the muscular twitchings in the face and lower limbs, which are seen in some cases of aortic regurgitation, are good examples of this group of symptoms.

(b) *Venous engorgement of distant organs and parts.*—The symptoms which are due to this cause are both numerous and important. In fact, many of the most prominent symptoms in cardiac cases are due to the secondary alterations in distant organs, and to the complications which result from the long-continued venous engorgements which I am now describing. *Venous engorgement of the lungs*, for example, produces shortness of breath, cough, hæmoptysis, etc., and predisposes to the attacks of bronchitis and pneumonia, which are so common in cardiac cases, and which are attended by characteristic symptoms. *Venous engorgement of the kidneys* produces marked alterations in the urinary secretion; the amount of urine is diminished; the specific gravity is high; the urine is highly coloured, loaded with urates, and often albuminous. In the course of time a form of cirrhosis of the kidney may be established.

In addition to these *lung and kidney* symptoms, others, due to engorgement of the *liver, stomach, intestines, brain*, etc., are commonly observed; while the *congestion of the subcutaneous veins, and the veins in the walls of the serous cavities*, produces dropsy, which is almost invariably first observed in the feet, but which may ultimately involve an extensive area of the subcutaneous cellular tissue and almost all the serous cavities of the body.¹ It is of great importance to remember that the occurrence of dropsy in these cardiac cases, depends in no

¹ Cardiac dropsy begins in the lower extremities, because being farther removed from the heart, the circulation in them is slower than in other parts of the body. It is generally worse at night, because during the day, when the patient is standing and going about, the return current of blood has to overcome the force of gravity in its passage backwards to the heart.

small degree upon the condition of the vaso-motor nerve tone, and the general vitality—the resisting power, so to speak, of the tissues. The composition of the blood, too, is an important element in its production.

In addition to the two great groups of symptoms already referred to, two minor groups may also be described :—

(a) In this group the symptoms are for the most part mechanical, but inasmuch as they are irregular and accidental in their occurrence, should be distinguished from the mechanical symptoms which I have hitherto described. To these symptoms, which are due to plugging of distant vessels by particles of fibrine (emboli) detached from clots in the heart, or from vegetations on the cardiac valves, the term *accidental* may be given.

The exact nature of these accidental symptoms depends for the most part upon the vessel which happens to be obstructed, upon the size of the plug, and upon the rapidity with which the obstruction is produced.

Particles of fibrine detached from the cavities or valves of the right heart produce embolic infarctions in the lungs, of which the chief symptom is hæmoptysis; limited inflammation of the pleura usually results, and, in those cases in which the infarction is of large size, the consolidation of the lung which it produces, can be recognised by percussion. Sudden obstruction of the main branches of the pulmonary artery—a condition which seldom occurs in cardiac cases, but which is sometimes seen after delivery, and in phlebitis,—may be followed by immediate death. In other cases, the termination is not so rapid, death being preceded by intense dyspnœa, cyanosis, quick and tumultuous action of the heart, rapid elevation of temperature, etc.

When the embolon is detached from the left cavities, it finds its way into some of the branches of the systemic arterial circulation. In some cases, it passes up the left common carotid artery, and lodges in the middle cerebral artery of the left side, producing right-sided hemiplegia and aphasia. In some of these cases the attack is ushered in by sudden loss

of consciousness, in others there are epileptiform convulsions; in others again (and more particularly in those in which the hemiplegia occurs more gradually) consciousness is retained.

In some cases the embolon lodges in the spleen; in others in the kidney; and, in fact, it may be carried to almost any part of the body.

The pathological character of the plug is also a point of some importance. In ordinary cases it consists, as I have already mentioned, of fibrine, and the effects which it produces are, for the most part, mechanical. In many cases of ulcerative endocarditis, micrococci abound in the vegetations covering the affected valves, and, being conveyed by the emboli to distant organs, there set up a similar infective process to that which is occurring in the heart.

(b) In this group the symptoms are the result of pressure. In intra-thoracic aneurisms, and in some cases of pericarditis, for instance, prominent symptoms, such as pain, dysphagia, alterations in the voice, cough, etc., may be due to this cause.

The more particular description of the symptoms, included in these four groups, will be more appropriately deferred until I come to treat of the individual cardiac diseases in detail. But it may perhaps be well, before going further, to consider a little more fully than we have yet done, dyspnœa, cough, and expectoration, three symptoms which are very common in cardiac cases, and which may be due to a considerable variety of different conditions.

Dyspnœa.

Alterations of the breathing are frequently met with in cardiac and arterial disease. In some cases, the difficulty of breathing is only occasional, in others, it is constantly present; in some it is due to over-exertion or other obvious exciting cause; in others, it occurs independently of any apparent external conditions. The chief forms of dyspnœa, and the conditions which give rise to them, are as follows:—

1. Breathlessness on exertion (going up stairs, climbing a hill, etc.), the breathing being natural when the patient is at perfect rest. This is the most common form of cardiac dyspnœa. It occurs more particularly in mitral lesions ; in those cases in which the right cavities of the heart are dilated ; and in cases of anæmia, in which, as we have seen, cardiac symptoms are common. (The dyspnœa in cases of anæmia is doubtless in great part due to the altered composition of the blood independently of the cardiac condition.)

2. The dyspnœa which results from alterations in position (probably from alterations in the position of the diaphragm) independently of pulmonary complications, such as bronchitis, œdema of the lungs and hydro-thorax. In many cases of advanced cardiac disease, more especially when the aortic arch, or the right cavities of the heart are much dilated, dyspnœa is readily excited by slight changes in position. It is common to find patients, breathing comfortably and quietly so long as they are sitting and at rest, unable to lie down for some time after getting into bed, on account of the dyspnœa produced by the alteration in their position. In some cases the heart, after a time, accommodates itself to the altered condition of things ; the breathing quiets down, and the patient falls asleep.

This form of dyspnœa is probably in many cases due to the altered position of the diaphragm, and seems closely allied to the difficulty in breathing, which cardiac patients so frequently experience after a full meal, when the stomach becomes distended with flatus, during attacks of dyspepsia, etc. In some cases it seems to be produced by reflex impressions passing from the stomach through the vagus nerve to the heart.

3. Paroxysmal attacks of dyspnœa. Sudden difficulty in breathing is occasionally met with in the course of cardiac affections. In some cases it is due to sudden over-distention of the cardiac cavities ; in others, to spasm of the glottis, produced by the pressure of an aneurism of the aortic arch upon the left recurrent laryngeal nerve ; in others, to the sudden pressure of an aneurism upon the

trachea; in others, to the sudden œdema of the lungs, which sometimes, though rarely, results from excessive over-distention of the left ventricle; in others to sudden distention of the right ventricle, and imperfect supply of blood to the lungs; while in exceptional cases severe dyspnœa may be caused by embolic plugging of the pulmonary artery.

In cases of this description the difficulty of breathing is intense; the patient has to sit up, and literally to fight for breath. The duration of the attack varies; in some cases, it is only temporary; in others (as for instance, in the case of an aneurism pressing upon the trachea or primary bronchi) it may continue for several days. In many cases death terminates the attack.

4. Typical Orthopnœa. In other cases the difficulty in breathing which was first slight, steadily increases, and a permanent condition of orthopnœa, which may continue for several days, or even weeks, is gradually developed. Such a condition is common in the late stages of cardiac cases, more particularly in advanced stages of mitral disease and in dilated conditions of the right heart. The patient is more or less cyanotic. Dropsy is usually a prominent symptom, and the difficulty in breathing depends, in many cases, upon pulmonary complications, such as hydro-thorax, bronchitis, œdema of the lungs, etc.

5. Cheyne-Stokes' Respiration: *Angina sine dolore*.—In some cases of cardiac disease, more particularly where the right heart is dilated and fatty, a peculiar form of rhythmical dyspnœa is observed, to which the term Cheyne-Stokes' respiration is given, after the two physicians (Drs Cheyne and Stokes) who first described it. In this form of dyspnœa the patient experiences considerable difficulty of breathing for a few respirations. The dyspnœa gradually subsides, the respirations becoming shallower and shallower, slower and slower, until for a time—it may be for half or three quarters of a minute—they are entirely suspended; they then gradually reappear, and become deeper and deeper, quicker and quicker, until the height of the paroxysm of dyspnœa is reached.

(See fig. 16.) The same sequence of events then recurs. The duration of the whole cycle from the height of one paroxysm of dyspnœa to the height of the next paroxysm, is usually from one to two minutes, half, or less than half, being occupied by the period of non-respiration or rest.

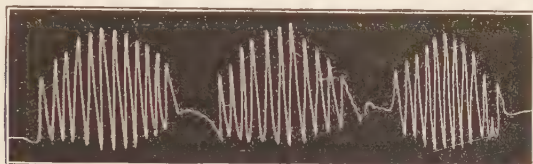


FIG. 16.—*Diagrammatic representation of Cheyne-Stokes' Respiration.*

Cheyne-Stokes' respiration is not pathognomonic of any single condition; it is a rare phenomenon, and has been observed in:—(a) advanced cases of cardiac disease (more especially, as I have already mentioned, in a dilated and fatty condition of the right heart, atheroma of the coronary arteries, and dilatation of the aorta); (b) certain cerebral affections (more especially lesions in the neighbourhood of the medulla oblongata); (c) uræmia; and (d) very exceptionally in other conditions.

Cheyne-Stokes' respiration is a very grave and ominous symptom; it only occurs in advanced cases of cardiac disease, and is, as a rule, speedily followed by death. It is closely allied to the condition which is termed *angina sine dolore*. In many cases of Cheyne-Stokes' respiration the arterial tension is very markedly increased; this was notably so in two typical cases which have come under my own observation. In some cases the condition continues for many hours, it may be for several days; the sufferings of the patient (though not painful in the common acceptance of the term) are extreme; he becomes much exhausted, and often very drowsy and sleepy. No sooner, however, does he dose off to sleep than he wakes with a horrid dream or with a start; and the waking is always associated with the period of dyspnœa, *i.e.* it always occurs, so far as my observation goes at the height of the paroxysm of dyspnœa.

In Cheyne-Stokes' respiration, then, there is an alternate condition of dyspnœa and apnœa, the two conditions being separated by a gradual rise and fall in the depth and frequency of the respirations.

The exact cause of the condition is not definitely known. Its immediate cause is, without doubt, perverted action of the respiratory centre in the medulla; and in order to understand the manner in which this perverted action may be brought about, I must now briefly describe the nervous mechanism of respiration. It seems probable:—

(1.) That the nerve impulses, which throw the respiratory muscles (the muscles of inspiration and expiration) into action, are discharged from a centre which is situated in the medulla oblongata—the so-called respiratory centre. (Respiratory centres are also situated in the spinal cord, but that in the medulla oblongata is certainly the most important, and seems to initiate the process, and to control the action of the lower, *i.e.* of the spinal respiratory centres.)

(2.) That under normal circumstances the action of this centre is rhythmical and automatic. (This point is not absolutely determined, but it seems probable that the action of the respiratory centre in the medulla is automatic.)

(3.) That although the action of this centre (the respiratory centre) is automatic, its discharges (both as regards their force and frequency) may be influenced by external conditions (*i.e.* conditions outside the centre); and that amongst these external conditions the amount of oxygen supplied to this centre by the blood is certainly the chief.

When the blood contains a small amount of oxygen (*i.e.* when it is highly venous), or when the arterial blood supply to the centre is diminished or abolished, its nerve discharges are increased, *i.e.* the force and (to a less extent) the frequency of the respiratory movements are increased; in other words, a condition of dyspnœa may be developed. The attacks of dyspnœa which occur in some cases of cirrhotic Bright's disease are probably due to this cause, *viz.*, to a diminished supply of arterial (oxygenated) blood to the respiratory centre. In chronic Bright's disease the arterial tension is

extremely high, in consequence, it is supposed, of a spasmodic contraction of the arterioles, and we can easily suppose that when the arterioles are tightly contracted, the blood supply to the respiratory centre in the medulla may be so much diminished as to produce a condition of dyspnœa. This form of dyspnœa is sometimes called renal asthma, the term is, however, a bad one, for the condition differs from the dyspnœa of spasmodic asthma in this important particular, that there is no obstruction to the passage of air into and out of the air cells. The respirations are not only more frequent, but they are much deeper than in health—to use the expressive words of my friend Dr Wyllic, a patient affected with this form of dyspnœa ‘looks as if he had just been running a mile race.’

When, on the contrary, the blood is over oxygenated, the nerve discharges from the respiratory centre are diminished in force and frequency, and a condition of apnœa may result.

A free supply of arterial blood to the medulla, therefore, produces diminution of the respiratory movements, while a diminished supply of arterial blood, or a venous condition of the blood, is attended with the opposite result.

The phenomena of Cheyne-Stokes' respiration are probably to be explained (in part at least) by the fact, that periodical variations occur in the amount of oxygen supplied to the respiratory centre in the medulla.

Various theories have been advanced to explain the manner in which Cheyne-Stokes' respiration is produced. None of them are, to my mind, perfectly satisfactory. The best known are those of Traube and Filehne.

Traube's Theory.—Traube, starting with the idea that in the different clinical conditions, which give rise to Cheyne-Stokes' respiration, there is a diminished supply of properly arterialised blood to the respiratory centre, supposed :—

(1) That in consequence of the deficient supply of oxygen, the irritability of the respiratory centre is so materially lowered, that a much larger accumulation than usual of carbonic acid in the blood is required to arouse it into action.

(2) That in order to provide this increased quantity of carbonic acid, the intervals between the different respiratory acts are necessarily prolonged, and that it is in this manner that the stage of apnoea is produced.

(3) That at the end of the period of apnoea, the accumulation of carbonic acid in the blood is so great, that the respiratory centre is aroused into action; that the stimulation is at first slow and imperfect, but that it gradually gains in intensity and strength until the discharge, which is excited by it, is so violent, that a condition of dyspnoea is produced.

(4) That in consequence of the increased respiratory efforts the blood becomes oxygenated; the respiratory centre is therefore no longer stimulated, and in consequence of the cessation of the stimulation, or possibly of the fact that its irritability is exhausted, the respiratory centre gradually ceases to discharge, and the period of apnoea supervenes.

Various objections may be urged against this view, but the main one—and it seems to me a fatal one—is this, that a diminished supply of properly arterialised blood to the respiratory centre would excite it to discharge, rather than lower its irritability. Venous blood stimulates the respiratory centre, not so much because it contains an excess of carbonic acid, as because it is deficient in oxygen. With regard to this point, Dr M. Foster says, ‘There can in fact be no doubt that the action of deficiently arterialised blood on the respiratory centre, as manifested in an augmentation of the respiratory explosions, is due primarily to a want of oxygen, and in a secondary manner only, to an excess of carbonic acid.’¹

Dr Sansom's Modification of Traube's Theory.—Dr Sansom agrees with the latter part of Traube's theory, but supposes that the impaired irritability of the respiratory centre, which Traube attributes to a deficient supply of sufficiently arterialised blood, is of a paralytic character, and that in many cases it is due to the direct result of a cerebral lesion. ‘I consider,’ he says, ‘that the initial lesion is paresis of the respiratory centre, and though this paresis *may be* produced by reflex nerve influence, it is usually a direct exhaustion from cerebral causes—once initiated, the explanation of the phenomena on the theory of Traube is complete.’²

With this opinion I cannot entirely agree. It is difficult to see how a centre, which is in a state of paresis or paralysis (unless we include under that head a condition of irritable weakness such as I believe to be at the root of the matter) can be excited to such vigorous action as the respiratory centre evidently is; and it is difficult, I think, to explain by this theory the total cessation of the respiration which occurs during the stage of apnoea. I differ from Dr Sansom in thinking that the irritability of the respiratory centre is impaired. It seems to me that a

¹ *A Text Book of Physiology*, 3d edition, p. 340.

² *Diagnosis of Diseases of the Heart*, p. 39.

condition of irritable weakness, in which it is *more irritable*, more easily excited to discharge, but at the same time *more easily exhausted* than in health, would more satisfactorily account for the phenomena. But to this point I will presently return.

Filehne's Theory.—Filehne believes that the vaso-motor centre, as well as the respiratory centre, is concerned in the production of the condition. He supposes :—

(1) That at the end of the period of apnœa the deficiency of oxygen and the excess of carbonic acid in the blood, stimulate the vaso-motor centre ; and that in consequence of this stimulation, the arteries throughout the body (including of course the cerebral vessels) are thrown into a condition of contraction.

(2) That in consequence of the diminished supply of arterial blood to the respiratory centre, which results from this arterial contraction, stimulation of that centre (the respiratory centre) occurs, and is manifested externally in the form of dyspnœa.

(3) That in consequence of the excessive respiratory efforts, the blood becomes quickly arterialised, the stimulation of the vaso-motor centre is thereby removed ; the tonic contraction of the arteries, therefore, disappears, and the respiratory centre is again supplied with arterial blood. Its discharges, therefore, become less and less powerful, and finally, when the arterial spasm is completely removed, the respiratory centre is so freely supplied with arterial blood that it no longer discharges, and the condition of apnœa is produced.

After the apnœa has continued for a longer or a shorter period, the blood again becomes venous, the vaso-motor centre is again stimulated, the arteries again contract, and the whole sequence of events is repeated.

This view at first sight seems very plausible, but it is certainly incomplete, unless we suppose that there is, in addition, some condition present which renders the discharges from the respiratory centre more easy at one period of the cycle (*i.e.* during the period of dyspnœa) and more difficult at the other period of the cycle (*i.e.* during the period of apnœa) than they are in health. It fails, I think, to account for the phenomena, unless we grant that some primary alteration in the respiratory centre, such as I have indicated, is present. If the conditions which Filehne suggests are all that are required, would not Cheyne-Stokes' respiration be of more frequent occurrence than it is? Would we not expect it to be produced in every case in which the blood is imperfectly aerated? Again, it fails, I think, to account satisfactorily for the apnœa. There are surely circumstances of every day occurrence in which the medulla is quite as freely supplied with arterial blood as it is in cases of Cheyne-Stokes' respiration during the period of apnœa, *i.e.* after the arterial spasm is relaxed ; and if Filehne's theory is correct, why is it that in these cases apnœa is not produced?

I am disposed, therefore, to think with Dr Sansom, that something more is necessary, and that there must be some alteration of the respiratory centre itself, in addition to the conditions which Filehne's theory supplies. A state of irritable weakness would, in my opinion, account for the condition.

The respiratory centre in the medulla oblongata probably consists of two parts—one connected with inspiration (the inspiratory centre), the other, with expiration (the expiratory centre). Now, according to Rosenthal (quoted by Dr M. Foster), the inspiratory centre is the seat of two conflicting forces, one tending to generate inspiratory impulses (the discharging portion of the inspiratory centre, as we may call it), and the other offering resistance to the generation of these impulses (the restraining or inhibiting portion of the inspiratory centre), the one and the other alternately gaining the victory, and thus leading to a rhythmical discharge.¹

Further, we may probably with truth suppose that the two parts of the inspiratory centre are differently acted upon by the same stimulus; venous blood, for instance, which excites the action of the discharging portion, depresses the action of the restraining portion, *vice versa* arterial blood depresses the action of the discharging portion, but strengthens the action of the restraining part.

Now, if we suppose that the discharging portion is in a condition of irritable weakness, in which it is more easily excited to discharge, but in which it tends to become more speedily and more completely exhausted than in health—(or, better still perhaps, that both portions of the centre are in this abnormal condition, *i.e.* a state of irritable weakness), we have, I conceive, a condition of things which will satisfactorily explain the phenomena.

Let us suppose, as it is simpler, a case in which the discharging portion is in a condition of irritable weakness, the restraining portion remaining normal. Starting, as we did in considering Filehne's theory, with the end of the period of apnœa, *i.e.* with the blood in a highly venous condition (see fig. 17), we may suppose:—

(1.) That the venous blood gradually excites a paroxysm of dyspnœa:—*Firstly* and chiefly by acting directly upon the inspiratory centre itself, depressing the action of the restraining portion, and arousing the action of the discharging portion, which has, during the stage of rest or apnœa, been gradually recovering from the condition of exhaustion occasioned by the excessive discharge, which produced the preceding paroxysm of dyspnœa. *Secondly*, by stimulating the action of the vaso-motor centre, in consequence of which the arterioles are contracted, and the supply of oxygen to the respiratory centre is still further diminished. (See fig. 18.)

¹ *Text Book of Physiology*, 3d edition, p. 336.

(2.) That in consequence of the *excessive irritability* of the discharging portion of the inspiratory centre, the discharges become excessive, and a condition of dyspnœa is produced. (See fig. 19.)

(3.) That in consequence of the *weakness* of the discharging portion of the inspiratory centre it speedily becomes exhausted—over exhausted; and the dyspnœa tends to subside. (See fig. 20.)

(4.) That in consequence of the excessive respiratory efforts during the paroxysm of dyspnœa, the blood (which was previously venous) becomes arterialised; stimulation of the discharging portion of the inspiratory centre ceases; stimulation of the restraining portion is produced; and in consequence of the deficient stimulation and over-exhaustion of the discharging portion, the restraining portion has full swing, and the condition of apnœa is produced. (See figs. 21 and 22.)

The arterialised blood acts *firstly* and chiefly upon the inspiratory centre itself, strengthening the action of the restraining portion and depressing the action (removing the stimulation) of the discharging portion; *secondly*, by removing the stimulation of the vaso-motor centre, in consequence of which the arterioles dilate, and the supply of oxygen (arterial blood) to the respiratory centre is still further increased.

During the stage of apnœa, the discharging portion, which was exhausted by excessive action during the period of dyspnœa, gradually regains its irritability, and the condition required for its stimulation, and for the removal of the control of the restraining portion, viz., a venous condition of the blood, is, in consequence of the absence of the respiratory movements, gradually developed.

In figures 17, 18, 19, 20, 21, and 22, I have endeavoured to represent the changes which I suppose occur in the different periods of the Cheyne-Stokes' cycle.

By this theory we can, I think, satisfactorily explain:—

(a) The occurrence not only of diminished respiratory movements after the period of dyspnœa, but the complete arrest of respiration which occurs during the stage of apnœa—a point which it is difficult to explain by the other theories.

(b) The remarkable fact that the respiratory centre is at one moment violently discharging, and at the next in a state of absolute quiescence.

(c) That the dyspnœa and apnœa follow one another with rhythmical regularity; and that the one condition gradually passes into the other, and *vice versâ*.

It is perhaps impossible in the present state of our knowledge to offer a decided opinion as to the manner in which the irritable weakness of the vaso-motor centre, which I have supposed is present in cases of Cheyne-Stokes' respiration, is produced. A deficient supply of arterial blood is probably in many cases one factor which aids in the production of the condition. We know that in conditions of profound anæmia, an extreme condition of irritable weakness of the spinal centres (in which the muscular

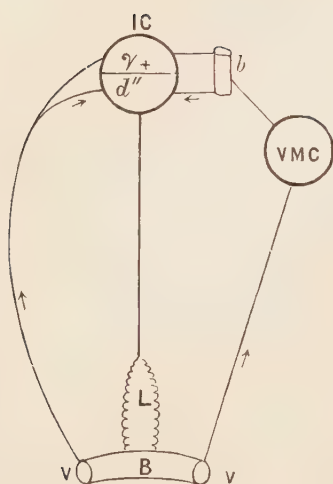


FIG. 17.

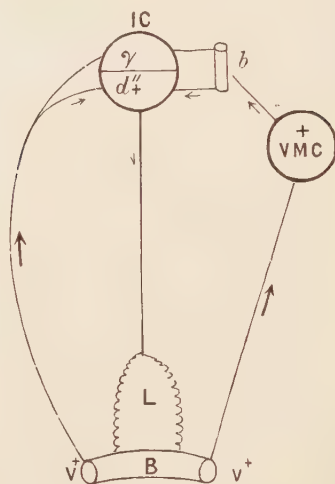


FIG. 18.

FIG. 17.—*The end of the period of apnoea.*

1. The discharging portion of the inspiratory centre is inactive but quite recovered from its previous exhaustion= d'' .
2. The restraining portion of the inspiratory centre (r) is still in excess= $r+$, but exhausted.
3. The lungs (L) are inactive.
4. The blood (B) is venous= $V+$; and a stimulus is passing:—to the discharging portion of the inspiratory centre (d''); and to the vaso-motor centre (VMC)
5. The vaso-motor centre is not acting.
6. The peripheral blood vessels (b) are dilated; and charged with venous blood; and in consequence of the absence of oxygen, the discharging portion of the inspiratory centre is being stimulated.

FIG. 18.—*The commencement of the period of dyspnoea.*

1. The discharging portion of the inspiratory centre is beginning to act, and has quite recovered from its exhaustion= $d''+$.
2. The restraining portion is not acting and is exhausted= r .
3. The lungs (L) are moderately distended=moderate dyspnoea.
4. The blood is highly venous ($V+$), and a stimulus is passing:—to the discharging portion of the inspiratory centre, and to the vaso-motor centre.
5. The vaso-motor centre is acting, the peripheral blood-vessels are moderately constricted, and charged with highly venous blood; a stimulus is consequently passing to the discharging portion of the inspiratory centre.

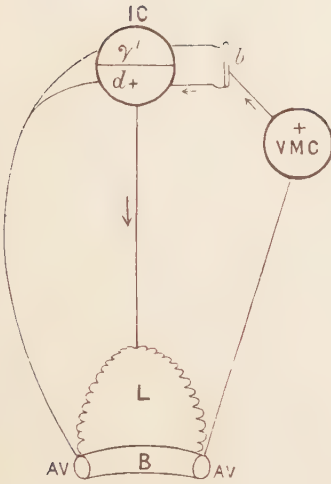


FIG. 19.

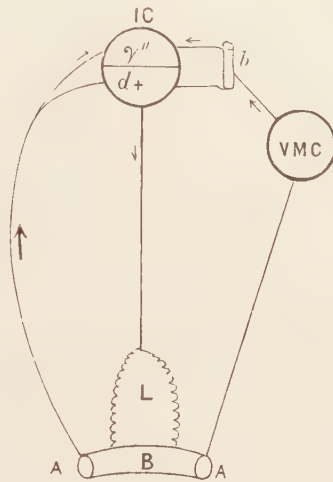


FIG. 20.

FIG. 19.—*The period of the height of the dyspnoea.*

1. The discharging portion of the inspiratory centre is acting very powerfully.
2. The restraining portion is inactive, but beginning to recover from its exhaustion.
3. The lungs (L) are acting very powerfully.
4. The blood is antero-venous=AV ; and not stimulating any of the centres.
5. The vaso-motor centre is discharging powerfully in consequence of the previous stimulation.
6. The peripheral blood-vessels are markedly contracted, and the discharging portion of the inspiratory centre is being stimulated by the want of oxygen.

FIG. 20.—*The end of the period of dyspnoea.*

1. The discharging portion of the inspiratory centre is acting feebly, and rapidly becoming exhausted.
2. The restraining portion has recovered from its exhaustion, but is not yet acting= r'' .
3. The lungs are acting feebly.
4. The blood is arterial=A, and a stimulus is beginning to pass to the restraining portion of the inspiratory centre.
5. The vaso-motor is still acting, though feebly.
6. The peripheral blood-vessels are moderately dilated, and charged with arterial blood ; a stimulus is consequently passing to the restraining portion of the inspiratory centre

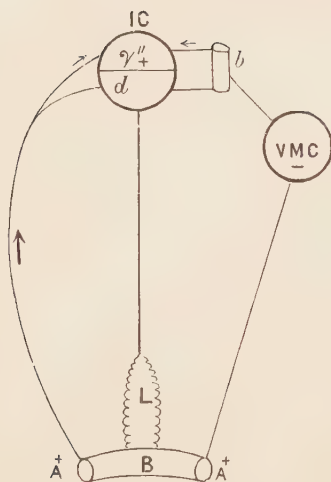


FIG. 21.

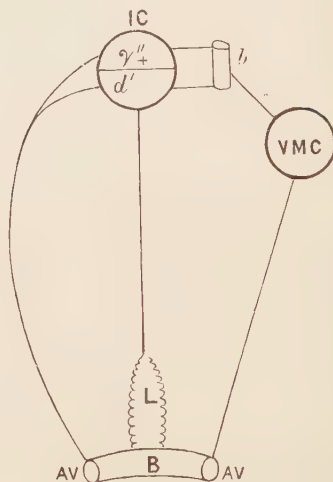


FIG. 22.

FIG. 21.—*The period of the commencement of apnoea.*

1. The discharging portion of the inspiratory centre is inactive and exhausted= d .
2. The restraining portion is acting, and has quite recovered from its previous exhaustion.
3. The lungs are inactive.
4. The blood is highly arterial ($A+$), and a stimulus is passing to the restraining portion of the inspiratory centre.
5. The vaso-motor centre is not acting.
6. The peripheral blood-vessels are dilated, and in consequence of the free supply of arterial blood the restraining portion of the inspiratory centre is being stimulated.

FIG. 22.—*The period of the height of the apnoea.*

1. The discharging portion of the inspiratory centre is inactive, but beginning to recover from its exhaustion= d' .
2. The restraining portion of the inspiratory centre is still acting powerfully= $r''+$.
3. The lungs are inactive.
4. The blood is arterio-venous (AV), and not stimulating any of the centres.
5. The vaso-motor centre is inactive.
6. The peripheral blood-vessels are widely dilated, and charged with blood, which is partly arterial and partly venous, and which stimulates neither portion of the inspiratory centre.

power is defective, muscular nutrition impaired, but muscular irritability both to direct and reflex stimulation notably increased) is often observed ; and in many cases of Cheyne-Stokes' respiration, the arteries are tightly contracted, the face is pale, and the supply of arterial blood to the nerve centres is deficient, *i.e.* a condition of local anæmia is present.

It is difficult, however, to suppose that a deficient supply of blood to the vaso-motor and respiratory centres is (even in these cases) the sole cause of the condition. And in those cases in which the arterial tension is not increased, and in which there is no anæmia, it is obvious that some other condition must be present.

Theoretically we may suppose that the condition (irritable weakness of the respiratory centre in the medulla) may be produced by :—

(*a.*) Lesions of the medulla itself.

(*b.*) Impressions passing to it from the higher nerve centres.

And, if these conditions occur, we can satisfactorily explain the mode of production of those cases of Cheyne-Stokes' respiration in which there are nervous lesions, and in which the heart is healthy.

(*c.*) Impressions passing to it from the periphery, more particularly from the heart and respiratory organs through the vagi and superior laryngeal nerves.

That the respiratory centre is powerfully affected through these channels, the passage quoted in the foot-note from Prof. M. Foster clearly shows ; and it is not difficult to conceive that in conditions of disease, profound modifications of the respiratory centre may in this manner be produced, more especially in those cases in which the right ventricle is affected, and the supply of blood to the lungs interfered with. Possibly then, a combined condition of defective arterial blood supply to the respiratory centre, such as is produced by a tonic contraction of the minute arteries, together with some abnormal stimulation through the vagi or superior laryngeal nerves,¹ such as we have supposed may be

¹ 'Among the afferent impulses which affect the automatic action of the centre' (*i.e.* of the respiratory centre) says Dr Michael Foster, 'the most important are those which ascend along the vagi. If one vagus be divided, the respiration becomes slower ; if both be divided, it becomes very slow, the pauses between expiration and inspiration being excessively prolonged. The character of the respiratory movement too is markedly changed ; each respiration is fuller and deeper, so much so indeed that, according to some observers, what is lost in rate is gained in extent, the amount of carbonic acid produced and oxygen consumed in a given period remaining after division of the nerves about the same as when these were intact. Without insisting too much on the exactness of this compensation, we may at least conclude from the effects of section of the vagi, in the first place, that during life afferent impulses are continually ascending the vagi and modifying the action of the respiratory centre, and in the second place, that the modification bears chiefly on the distribution in time of the afferent respiratory impulses, and not so much on the amount to which they are generated. These

produced by the cardiac lesion (more particularly by a dilated condition of the right heart), may be the cause of the Cheyne-Stokes' respiration which is met with in some cardiac cases.

The whole subject (*i.e.* Cheyne-Stokes' respiration) requires further elucidation. It is only by the accurate examination, and by the publication of a considerable number of cases that we can hope to arrive at a satisfactory conclusion as to the exact nature of the condition. In order to facilitate the study of these cases I have drawn up the following memorandum showing the points to which attention should be particularly directed in the clinical examination.

The points to which attention is to be directed in the clinical examination of Cheyne-Stokes' respiration.

A. *Preliminary facts.*—Name—Age—Sex—Occupation—Address—Date of Examination.

B. *History.*

(1.) Of the present illness.—The date of commencement; the exact mode of commencement; the supposed cause of the attack; the character of the symptoms; the order of their appearance; and the treatment which has been adopted, up to the date of the observation.

(2.) The health history prior to the commencement of the present attack.

(3.) The family history; especially the occurrence of disease of the heart or kidneys; and of attacks of Cheyne-Stokes' respiration amongst near relatives.

C. *The present condition.*

(1.) The nature of the primary (main) disease.

(2.) The condition :—(a) of the heart (which should be carefully and methodically examined in accordance with the plan of examination

afferent impulses are probably started in the lungs by the condition of the blood in the pulmonary capillaries acting as a stimulus to the peripheral endings of the nerves, though possibly the altered air in the air cells may also act as a stimulus to the nerve endings.' . . . Again, 'if the central end of the superior laryngeal branch of the vagus be stimulated, whether the main trunk of the nerve be severed or not, a slowing of the respiration takes place, and this may, by proper stimulation, be carried so far that a complete standstill of respiration in the phase of rest is brought about, *i.e.* the respiratory apparatus remains in the condition which obtains at the close of an ordinary expiration, the diaphragm being completely relaxed. In other words, the superior laryngeal nerve contains fibres, the stimulation of which produces afferent impulses, whose effect is to inhibit the action of the respiratory centre; while the main trunk of the vagus contains fibres, the stimulation of which produces afferent impulses, whose effect is to accelerate or augment the action of the respiratory centre.'¹—*A Text-Book of Physiology*, fourth edition, pp. 356, 357.

previously described, see page 58); (*b*) of the lungs; (*c*) of the kidneys (amount of urine in twenty-four hours, its colour, reaction, specific gravity, presence or absence of albumen and casts); (*d*) of the nerve centres.

(3.) The appearance of the patient during the attack of Cheyne-Stokes' respiration, and the sensations which he experiences.

(4.) The exact character of the respiration itself. The exact duration of the whole cycle; the exact duration of the period of dyspnœa; the number and character (depth, etc.) of the respirations during the period of dyspnœa; and the exact duration of the period of apnœa. The observer should endeavour to represent diagrammatically on paper the character of the respirations during the whole cycle; preserving, so far as possible, the relative frequency and depth of the respiratory movements.

(5.) The condition of the peripheral circulation. The colour of the face, during the periods of dyspnœa and apnœa, respectively. The frequency of the pulse during the periods of dyspnœa and apnœa. The tension of the pulse during the periods of dyspnœa and apnœa. In all cases sphygmographic tracings should be carefully taken both during the periods of dyspnœa and apnœa, and, if possible, a continuous tracing during the whole cycle, *i.e.* from the commencement of one period of dyspnœa to the commencement of another period of dyspnœa should be obtained.

(6.) The treatment adopted and its effects; in particular, the effect of nitrite of amyl inhalations, or of nitro-glycerine.

(7.) The result.

(8.) In fatal cases the record of the *post-mortem* examination.

Cough and Expectoration.

Cough is a common symptom in cases of cardiac disease. It generally depends upon venous engorgement of the lungs or upon the secondary pulmonary complications, such as bronchitis, œdema of the lungs, pneumonia, etc., which are, as we have already seen, of very frequent occurrence. In cases of this description, the cough is usually moist, the character of the expectoration varying with the nature of the pulmonary complication. In other cases, the cough is due to reflex irritation, it is then usually dry, and often has a harsh, brassy, clanging character. Aneurisms or simple dilatations of the aortic arch, which exert irritative pressure upon the recurrent laryngeal nerve, are attended by a characteristic cough of this description. A short, dry cough is also seen in

some cardiac cases (in which there are no distinct pulmonary complications, and in which there is no pressure upon the recurrent laryngeal nerve), and is possibly produced by reflex irritation of the terminal branches of the cardiac nerves in the walls of the heart itself.

Expectoration.

The character of the expectoration depends chiefly upon the exact nature of the pulmonary complication which is present. In acute bronchitis, for example, the expectorated matters are white and frothy, and consist chiefly of watery mucus; in chronic bronchitis the expectoration is mucopurulent or purulent; in pneumonia it is scanty and viscid, and may present the characteristic rusty-red colour; in very acute oedema of the lungs, a large quantity of liquid is suddenly poured out into the air-cells and minute bronchi, there is great dyspnœa, both inspiration and expiration may be attended with a loud roaring stridor, and the patient may expectorate a large quantity of frothy fluid, which in some cases is highly albuminous. Hæmoptysis is not unfrequent in the course of cardiac cases. In some it depends upon simple engorgement of the lungs, and the rupture of dilated capillaries in the walls of the air cells. In this form of hæmoptysis, which is common in mitral stenosis, the blood may be expectorated in considerable quantities, and is often quite pure. In other cases hæmoptysis depends upon embolic plugging of some of the pulmonary vessels (pulmonary infarctions); in cases of this description the blood (which may at first be pure) soon becomes of a dark colour, and is usually mixed with considerable quantities of mucus; as the case progresses, the expectoration may assume various shades, and may become fœtid. In other cases again, hæmoptysis depends upon the rupture of an aneurism into the air passages. In cases of this description, the blood is bright and florid, and the bleeding may be sufficiently copious to be followed by immediate death.

The comparative significance of 'subjective cardiac sensations,' and of symptoms indicative of mechanical derangement of the circulation.

In many cases of functional derangement of the heart, more especially in the great group of neurotic lesions, there are no 'mechanical' symptoms, but the patient complains *only* of 'subjective cardiac sensations.' He often, too, is impressed with the belief that he has heart disease, and comes to the physician with the express object of having his heart examined.

Persons suffering from organic cardiac disease, on the contrary, *usually* complain of the lung, stomach, and other symptoms, which result from mechanical derangement of the circulation, though they may in addition experience and complain of 'subjective cardiac sensations.' The practical lesson to be derived from these facts is, that persons who come to a physician complaining of their hearts, and *who do not manifest any mechanical disturbance of the circulation*, are in all probability free from serious cardiac disease;¹ while in those cardiac cases in which such symptoms as cough, shortness of breath, dropsy, etc., are present, as the result of the cardiac lesion, there is some structural alteration which has produced mechanical derangement of the circulation; occasionally, as in anæmia, this structural alteration is temporary and curable, but in most cases it is permanent and 'organic.' The reader must not, of course, conclude from this statement, that the converse proposition holds good, viz., that all persons who have an organic cardiac lesion, manifest well-marked symptoms. Such is not the case, for, as we have previously seen, many serious organic lesions are for a long time latent (*i.e.* are unattended by symptoms), being balanced by the compensatory changes which occur.

¹ True angina pectoris is a notable exception to this statement. In that condition intense pain, commencing in the region of the heart, and radiating up to the left shoulder and down the left arm, is experienced. The pain is attended with a dread of impending death, but there are often no other symptoms indicative of serious organic disease.

The History of the Case.

History of the present illness.—In the first place, it is important, if possible, to ascertain the *exact date at which the symptoms*, for which the patient comes under observation, *commenced*. By determining this point we can, provided that he has been previously healthy, ascertain with considerable probability whether the cardiac lesion is a recent one or not.¹

In some cases the point is easily determined. A patient affected with aneurism, for example, may tell you that, while making some violent exertion, he felt something give way in the chest or abdomen, and that his symptoms date from that event. In cases of acute rheumatic inflammation of the heart, there is usually no difficulty in fixing the exact date at which the rheumatic attack commenced. So, too, in some functional derangements, more especially in some forms of neurotic palpitation and the like, the attack comes on suddenly and abruptly, the patient having been previously free from any cardiac symptoms. But in other cases, more especially in the great group of chronic cardiac affections, the symptoms develop so gradually, and the case progresses so slowly, that the patient is unable to fix a definite date for the commencement of his illness.

In the second place, the *exact mode of commencement of the attack, the character of the symptoms, and the manner in which they are progressing*, should be ascertained. The exact character of any apparent cause, such as violent effort, mental agitation, an attack of rheumatism or scarlet fever, should be carefully investigated.

In the third place, it is important to ascertain *the nature of the treatment which has been adopted up to the time when the patient comes under observation*. Digitalis, for example, when given in full doses, may materially modify the action of the heart; the knowledge, therefore, that the patient had been taking large doses of digitalis, might be a point of great

¹ The fact that the symptoms are of recent origin is not proof positive that the lesion is a recent one, for grave cardiac lesions may, as I have repeatedly pointed out, be, for a time at least, unattended by any obvious symptoms.

practical importance. In other words, the nature of the treatment which has been previously adopted, may give us a clew to the nature of the case, and indicate the opinion of the previous medical attendant.

The history of the state of health before the commencement of the present illness, is a point of the greatest practical importance.

The patient should always be closely questioned as to the occurrence of previous attacks of rheumatism,¹ more especially of rheumatic fever. When a history of acute rheumatism is elicited, it is important to ascertain if the heart was affected during the attack. The fact that the patient was leeches, cupped, or blistered over the præcordia, is important evidence in those cases in which he is unable to give us any definite information on this point. That the patient has been short of breath on exertion (on going up stairs, up a hill, etc.), since the attack of rheumatism, is also highly suggestive of a chronic valvular lesion, although he may have in other respects enjoyed good health. Scarlet fever is another disease, which is, not unfrequently, attended with endocarditis; and in children, who come under treatment for valvular lesions, it is important (more especially where there is no history of rheumatism) to inquire into this point. I might mention many other conditions which sow the seeds of subsequent cardiac or arterial disease (syphilis, for example, is an important cause of aneurism), they will however be detailed when I come to speak of the ætiology of the individual cardiac diseases.

The habits and mode of life, and general surroundings of the patient, are of great importance, more especially in determining the plan of treatment: but these points will be more appropriately considered afterwards.

¹ It is important to remember that endocarditis may develop in the course of mild and apparently insignificant attacks of subacute rheumatism. Many persons, too, more especially in the lower ranks of life, do not appreciate the difference between ordinary muscular rheumatism and rheumatic fever (articular rheumatism.) It is well, therefore, in the first instance to ask the patient if he has suffered from rheumatism, and if he answer in the affirmative, to question him as to the exact nature of the attack

Family History.—Acute rheumatism, which is such a fertile source of cardiac valvular lesions, is much more frequent in some families than in others ; and it is in consequence of their liability to rheumatism, that the members of these families are more apt to be affected with cardiac valvular lesions than other people. But, independently of the rheumatic tendency, some persons inherit a tendency to disease of the heart and vascular system. For example, the late Dr Charlton of Newcastle-on-Tyne, told me, that two brothers, in easy circumstances, who had not been exposed to any undue strain, and who had not suffered from rheumatism, came under his care, suffering from atheroma of the thoracic aorta, and died at the unusually early ages (for atheroma) of twenty-three and twenty-five. In these cases it was difficult to resist the conclusion that the condition was hereditary. In later life, when arterial degeneration is common, this hereditary tendency to atheroma is still more apparent. In some cases it seems to be due to gout ; in others it is associated with kidney disease. Those functional forms of cardiac derangement, which depend upon disordered innervation of the heart, are more common in persons of a neurotic temperament than in other people ; and as we all know the neurotic temperament is eminently hereditary.

PRESENT CONDITION.

[The date at which the examination is made should be stated, for in hospital practice several days may elapse between the admission of the patient and the noting of the case.]

The Physiognomy of the Case.—While the preliminary facts and previous history are being investigated, the physician is both consciously and unconsciously learning many important particulars as to the nature of the case.

In some cases the physiognomy not only suggests the nature of the lesion, but also indicates its severity. Attention must be particularly directed to :—

1. The colour of the face (of the lips, nose, and ears more especially).

2. The presence of subcutaneous dropsy (in the feet, face, and hands more particularly.)
3. The facial expression.
4. The condition of the breathing.
5. The attitude.
6. The general state of nutrition.

The Colour of the Face.

By observing the colour of the face we get important evidence as to the condition of the capillary circulation; and hence as to the manner in which the circulation is being carried on—a point of the greatest importance in cases of cardiac disease. In some cases the colour of the face is natural; in others it is more dusky than in health; in others, again, it is paler than normal.

Natural Colour of the Face.—In many functional affections, more particularly the neurotic disorders of the heart; in many aneurisms; in aortic stenosis; in slight cases of aortic regurgitation; and in mitral cases (more especially mitral stenosis), so long as compensation is perfect, the colour of the face may be quite natural.

Blueness of the face—Cyanosis.—Anything which interferes with the circulation of the blood in the lungs, with the passage of the blood through the right cavities of the heart, or which prevents the return of blood to the heart, such as pressure on the superior cava, will produce venous congestion, and hence blueness of the face. The cyanosis is most marked in those cases in which there is a backwash through the tricuspid, or in which the venous and arterial blood currents are intermixed, as they are in some congenital malformations.

Congenital Cyanosis, in which the blueness is often extreme (hence the term *morbis ceruleus*, which has been given to these cases), usually depends upon some cardiac malformation, such as stenosis of the orifice of the pulmonary artery with a patent condition of the foramen ovale, or upon transposition of the aorta and pulmonary artery (the aorta arising from the right, and the pulmonary artery from the left ventricle).

Acquired Cyanosis.—When the cyanosis is extreme, there

is generally, in my experience, long standing disease of the lungs or disease of the right cavities of the heart, permitting of tricuspid regurgitation: the two conditions being generally combined. Cases are, for instance, not unfrequently met with in which there is a history of repeated attacks of bronchitis since childhood; in which the patient has for years been short of breath; in which the lungs are emphysematous or cirrhotic; and in which the right cavities of the heart are hypertrophied and dilated. In cases of this description, there is always more or less blueness of the lips, and any increased obstruction to the aeration of the blood in the lungs (such as is occasioned by an attack of acute bronchitis, pneumonia, etc.), or any increased distention of the right cavities of the heart, may be attended with the most extreme degree of cyanosis.

In mitral cases in which, as we have previously seen, the free circulation through the lungs is interfered with, and in which secondary changes in the right heart are so commonly observed, more or less cyanosis is usually present, but it is seldom so great as in the cases of primary pulmonary obstruction to which I have just alluded, and is often associated with some degree of anæmia. While compensation is perfect the colour of the face may be quite natural.

In all of these cases, but less so, I think, in congenital cyanosis than in the other two, the face may be somewhat swollen as well as cyanotic.

When the return current of blood through the superior vena cava is interfered with, as it is sometimes by the pressure of an aneurism or solid intra-thoracic growth, the lips, ears, nose, etc., are more or less livid. In these cases there is marked œdema, which is not, of course, confined to the face, but is seen in all the parts (face, neck, upper extremities, and upper part of the thoracic parieties) from which the superior cava draws its blood-supply. In consequence of the œdema, the face in the region of the eyelids, etc., is usually pale, and the condition may at first sight be mistaken for a case of Bright's disease. The marked lividity of the lips, ears, etc., and the limitation of the swelling to the area of distribution of the superior cava, at once, distinguish the two conditions.

Pallor of the Face.—In cases of free *aortic regurgitation*, in which the arterial system is imperfectly distended during the diastole of the ventricle, the face is pale, generally thin, and there is often an anxious expression of countenance.¹

In conditions of *anæmia*, in which, as we have previously seen, cardiac symptoms and cardiac murmurs are common, the face and mucous membranes are unusually pale; in *chlorosis* and *progressive pernicious anæmia*, the face may be slightly puffy, and generally has a lemon yellow hue, which must be distinguished from the dingy, yellow colour, which is seen in many cases of advanced mitral disease.²

Cardiac lesions are, of course, frequently met with in connection with Bright's disease; and in combined cases of cardiac and renal disease, the pale, puffy face which is characteristic of the large white kidney, or the dingy colour of the face which is seen in the cirrhotic form of Bright's disease, may, of course, be met with.

The presence of subcutaneous dropsy.

As we have previously seen, dropsy is of frequent occurrence in advanced cases of cardiac disease (more especially mitral and right sided lesions); and it almost invariably commences in the feet.³ I have already alluded to those cases of

¹ This statement does not, of course, apply to those cases of aortic regurgitation in which the mitral valve has given way. In combined cases of this description, more particularly when the mitral regurgitation is free, there may be some blueness of the lips.

² In both of these cases the conjunctiva may be yellow. In the former (*chlorosis*) the yellow colour is seen chiefly at the inner and outer canthi, and is due to a deposit of sub-conjunctival fat; in the latter (advanced cases of mitral disease, tricuspid regurgitation, etc.) the yellow discolouration of the skin is of a darker, dingier hue, and usually depends upon congestion of the liver.

³ A swollen condition of the feet, untied shoes, slit-up trousers, etc., at once suggest a cardiac lesion. The same form of dropsy (*i.e.* dropsy beginning in the feet) may be due to simple debility, to anything which interferes with the return current through the inferior vena cava, or to primary obstruction to the blood in the lungs, such as is produced by cirrhosis, emphysema, etc. The differential diagnosis which can generally be made without much difficulty, will be more particularly described under the head of valvular lesions.

local dropsy (dropsy confined to the face, neck, and upper extremities) due to the pressure of an aneurism or intra-thoracic growth upon the superior cava.

The Facial Expression.

The facial expression (irrespective of the colour of the face and the presence or absence of œdema) does not, as a rule, afford much information. In exophthalmic goitre, in which cardiac symptoms and signs are usually prominent, the projecting eyeballs at once attract attention. In some cases of aortic disease, or of aneurism, the expression may be anxious and indicative of suffering. During the paroxysm of angina pectoris, in conditions of orthopnœa, and Cheyne-Stokes' respiration, an expression of horror, dread, anguish, or intense suffering, may be present. In advanced conditions of cyanosis, in which the cerebral centres are imperfectly supplied with arterial blood, the patient is often drowsy and the expression apathetic.

The Condition of the Breathing.

In cases of aortic disease there may be no disturbance of the respiration. In other cases of cardiac disease, one or other of the different forms of dyspnœa, which I have previously described (see page 66), may be present.

The Attitude.

In acute cases the patient is usually in bed, not so much on account of the cardiac complication as on account of the primary affection (acute rheumatism, etc.); he usually lies on his back, and, so far as possible, avoids movement.

In those cases of cardiac disease, both acute and chronic, in which the respiratory functions are seriously interfered with, the patient is unable to lie down, and a condition of typical orthopnœa may be present. Frequently he will not go to bed, but sits up in a chair for days and nights—it may be for weeks—together. The position, in which he places

himself in order to get relief, is sometimes a striking one ; when he is sitting in a chair or on the side of the bed, the head is bent forwards, the weight of the trunk being supported by the arms, which are kept rigid, the hands grasping the thighs just above the knees ; in this position the parts are so fixed that the respiratory muscles, more particularly the extraordinary muscles of respiration, are able to act with great advantage ; in other cases, he leans over the back of a chair, or kneels on the floor, resting his head and forearms on the bed, or sits resting them on a table, mantelpiece, etc.

Patients suffering from aneurism sometimes assume peculiar positions in order to remove the pressure of the sac from the surrounding parts ; in some cases of abdominal aneurism, for example, the patient can only lie on his face ; in others, he can only get relief from paroxysms of pain by resting on his hands and knees ; in others, again, the thigh is flexed upon the abdomen. But more minute and detailed description of the various attitudes which may be assumed in individual cases, would obviously be out of place here.

The General State of Nutrition.

The cardiac affections which seriously embarrass the respiration, and cause secondary disturbances in the liver, stomach, and other abdominal organs, are usually attended with very considerable derangement of the nutritive functions. In mitral cases, for example, after the failure of compensation, the patient becomes soft and flabby, and there is usually considerable emaciation ; it must of course be remembered that when the subcutaneous fat is abundant, or there is great dropsy, the emaciation may escape detection unless carefully looked for.

In aortic cases the patient is usually spare, but the general state of nutrition is, as a rule, fairly well preserved.

In some cases of aneurism there is no impairment of the general health ; indeed one of the most striking features is often the total want of proportion, so to speak, between the severity of the disease and the general constitutional state.

THE PHYSICAL EXAMINATION OF THE HEART AND
ORGANS OF CIRCULATION.

The object of the physical examination of the heart and organs of circulation is, of course, to ascertain their exact physical condition. We endeavour to determine the physical condition of the heart itself; of the great blood-vessels within the thorax and abdomen; and the manner in which the peripheral circulation is being carried on. The methods of examination which we chiefly¹ employ are inspection, palpation, percussion, auscultation, and the use of the sphygmograph.

THE PHYSICAL EXAMINATION OF THE HEART.

The object of the physical examination of the heart is to determine—

1. *The exact frequency, rhythm, and character (whether slow and laboured, quick and irritable, etc.) of its contractions.*

These points are determined by observing the character of the pulse; by inspecting and palpating the præcordia; and by auscultating the heart; the examination of the pulse being especially valuable.

2. *Its position in the thorax; its size and shape as a whole; and the relative size and shape of its component parts.*

We determine these points chiefly by inspecting the præcordial region (*i.e.* by observing by means of inspection the position, extent, and character of the visible impulse); by palpating the præcordial region (*i.e.* by observing by means of palpation the position, extent, and character of the visible impulse); and by percussing the præcordial region (*i.e.* by determining by means of percussion the exact extent, shape, and outline of the cardiac dulness).

Percussion is particularly valuable in determining these points.

¹ The cardiograph is not yet employed, even in hospitals, as one of the ordinary methods of examination; it is not, therefore, described in the text, but in the appendix.

3. *The condition of its valvular apparatus.*

The condition of the valvular apparatus of the heart is chiefly determined by means of auscultation. Palpation in some cases also affords information. The exact size and shape of the heart and of its component cavities (which are, as we have just seen, chiefly determined by means of percussion), corroborate the information derived from auscultation; while very important evidence as to the condition of the valvular mechanism, is also obtained by observing the condition of the circulation in front of and behind the valve which is being examined. The mode of examination will afterwards be described.

4. *The state of its muscular walls* (whether hypertrophied, dilated, degenerated, etc.).

We determine this most important point by observing:—

(a) The size of the heart and of its component parts (inspection, palpation, and percussion of the præcordia).

(b) The force and character of the cardiac impulse (palpation of the præcordia); the force and character of the pulse; the loudness and other characters of the heart sounds (auscultation of the præcordia), and the condition of the peripheral—arterial and venous—circulation. Corroborative evidence is also afforded in many cases by observing the general condition of the system, and the state of particular organs, such as the lungs and kidney: profound anæmia, for example, suggests fatty degeneration of the heart; kidney disease (especially cirrhosis), hypertrophy of the left ventricle.

5. *The condition of the pericardial sac.*

This point is chiefly determined by means of auscultation and percussion.

INSPECTION AS APPLIED TO THE EXAMINATION OF THE HEART; INSPECTION OF THE PRÆCORDIAL REGION.

By the præcordial region we understand that part of the chest wall which lies in front of the heart. It is necessary, therefore, before proceeding further, to define the exact position of the heart in the thorax, and to describe the limits of the præcordial region.

The position of the Heart in the Thorax.

The heart, contained in the fibro-serous bag termed the pericardium, is placed obliquely, with its long axis from right to left, in the lower part of the anterior mediastinum (see fig. 23). It consists of four chambers, two auricles and two

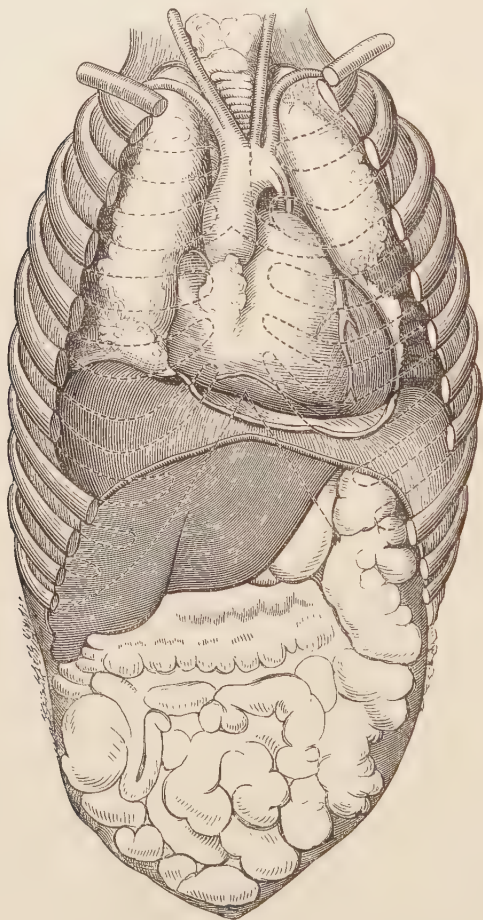


FIG. 23.—*The heart and great vessels in situ.* (Enlarged from Sibson.)

The lungs have been drawn aside, and the anterior surface of the pericardial sac removed.

ventricles ; and in the adult male, after death, is about the size of the closed fist. It rests upon the central tendon of the diaphragm, is attached above by its base to the great vessels, and is almost completely surrounded by the lungs, which, with the bony walls of the chest, protect it from external injury.

The *base* corresponds to the junction of the upper margins of the third costal cartilages ; the *apex* to the 5th interspace, or the 6th rib. (In the adult male the apex-beat can usually be felt between the 5th and 6th ribs, at a point about an inch and a half below, and slightly within the left nipple.) *As a whole*, then, the heart extends *vertically* from the 2nd interspace above, to the 6th rib below ; and *transversely*, from a little within the left nipple, to a finger's breadth or more to the right of the sternum ; and this space which the heart occupies, *as a whole*, is called the *deep cardiac region*.

Nearly two-thirds of the organ lie to the left, and more than one-third to the right of the middle line. Sibson points out that when the left lung is unusually large, *i.e.* larger than the right, the heart is situated more to the right ; and *vice versa*, when the right lung is larger than normal (irrespective, of course, of any disease, for we are now considering the position in health), the heart may be situated more to the left.

The relationship of the Heart to the surface of the Chest.

The præcordial region (that part of the chest wall which lies in front of the heart) includes, therefore, the greater part of the lower sternal and left mammary, and part of the right mammary, regions. (See fig. 24.)

The anterior surface of the chest, and the root of the neck, have for facility of description and for clinical convenience, been artificially divided into the following regions :—

The supra-sternal, upper sternal, and lower sternal regions, in the middle line. The supra-clavicular, clavicular, infra-clavicular, mammary and infra-mammary regions, on each side.

The boundaries of these regions are as follows :—

The supra-sternal region (1. fig. 24) is situated immediately above the upper end of the sternum, and is bounded on each side by the anterior borders of the sterno-mastoid muscles ; its upper boundary corresponds to the upper end of the trachea.

The upper sternal region (2. fig. 24) comprises the part of the chest which is situated behind the upper portion of the sternum. The upper boundary of this region is represented on the surface of the chest by the upper end of the sternum, the lower end by a line drawn across the sternum at the level of the junction of the third costal cartilages ; laterally this region is bounded on each side by the corresponding edge of the sternum.

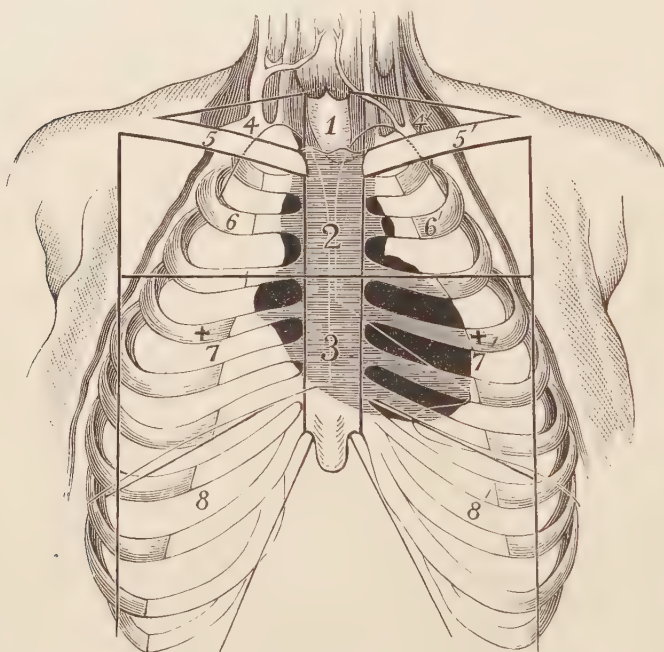


FIG. 24.—*The regions on the front of the Chest.* (Modified after Walshe.)

1. Supra sternal region.
2. Upper sternal region.
3. Lower sternal region.
- 4,4'. Right and left supra-clavicular regions.
- 5,5'. Right and left clavicular regions.
- 6,6'. Right and left infra-clavicular regions.
- 7,7'. Right and left mammary regions.
- 8,8'. Right and left infra-mammary regions.

Relationship of the Heart to the Chest Wall. 97

The lower sternal region (3, fig. 24) corresponds to the lower portion of the sternum.

The supra-clavicular region (4 and 4', fig. 24) is triangular in shape, and is situated immediately above the clavicle; on its inner side it is bounded by the outer side of the supra-sternal region, below by the clavicle; its third side being formed by an artificial line drawn from the outer end of the clavicle to the upper end of the trachea.

The clavicular region (5 and 5', fig. 24) corresponds to the inner half of the clavicle.

The infra-clavicular region (6 and 6', fig. 24) is bounded above by the clavicle; below by a line drawn across the chest on the level of the third rib; internally by the edge of the sternum; externally by an imaginary line falling vertically from the inner edge of the acromial process.

The mammary region (7 and 7', fig. 24) is situated immediately below the infra-clavicular region; and is bounded below by a slanting line drawn through the middle of the sixth costal cartilage; internally by the edge of the sternum; and externally by the vertical 'acromial line' described above.

The infra-mammary region (8 and 8', fig. 24) is situated immediately below the mammary region; its lower margin corresponds to the lower margins of the false ribs; internally it is bounded by the edge of the sternum, and below that bone by the margins of the ribs; externally by the 'acromial line.'

The exact relationship of the cardiac cavities to the front of the chest is, according to Walshe, as follows:—The anterior surface of the heart is chiefly formed by the right cavities; the tip of the left auricular appendix, and a narrow strip of the left ventricle being the only parts of the left heart, which are visible when the anterior surface is exposed *in situ*.

The *right auricle* reaches into the right mammary region, mainly on the level of the third cartilage and fourth interspace, slightly on that of the second space.

The *right ventricle* corresponds mainly to the lower sternal and left mammary regions, its inferior and nearly horizontal border stretching from the fifth right cartilage to the point at which the apex beats.

The *left auricle* lies deeply behind the root of the pulmonary artery, little but the appendix being visible.

The *left ventricle* extends vertically from the third to the upper edge of the sixth left costal cartilage, and occupies a portion of the left mammary region—a comparatively narrow strip being visible anteriorly.

The entire of the left ventricle, the greater part of the left auricle, and a large portion of the right ventricle towards the apex, lie to the left of the sternum. On the level of the fourth cartilage the widths of the heart substance lying on either side of the left border of the sternum are very closely the same.¹

¹ *Diseases of the Heart*, p. 4.

Method of inspecting the Chest.

The patient should be stripped to the waist, and placed directly opposite the observer, in a good light, in a sitting or semi-recumbent position. In cases of suspected aneurism, in which it is important to note the slightest elevation of the chest-wall above the level of surrounding parts, the suspected region of the chest must be well illuminated, and the observer having placed himself on the opposite side of the patient to that from which the rays of light are proceeding, should bring his eye to the same horizontal plane as the region of the chest which he wishes to examine. In this manner slight local elevations can be most readily observed.

The points to be observed in inspecting the præcordial region are :

1. Its form and conformation.
2. The position, extent, and character of the visible impulse, especially the position of the apex beat.
3. The condition of the integument. (This is a point of comparatively little importance).

The form and conformation of the præcordial region.

In health the two sides of the chest are practically symmetrical, and the left mammary region does not differ in form and shape from the corresponding region on the opposite side.

Alterations in the form and conformation of the præcordial region are frequently met with in disease ; in some cases it is unduly prominent, in others it is flattened or retracted.

Undue prominence of the præcordial region.

This condition may be due to :

1. Congenital or acquired alterations in the shape of the thorax, such as are seen in some cases of rickets, or in connection with some curvatures of the spinal column.

Diagnosis.—In these cases the character of the prominence ; the presence of other associated alterations in the bony walls of the thorax, such as a well-marked spinal curvature ; and the history of the case, enable us without difficulty to determine the cause of the prominence.

2. Abnormal conditions of the thoracic parieties in the præcordial region, such as an abscess in the subcutaneous cellular tissue, or an exostosis, or other new formation springing from the ribs or cartilages.

Diagnosis.—In these cases—which are rare—the diagnosis presents no difficulty.

In the case of an *abscess in the chest wall* there is tenderness on pressure over the swelling, and often redness of the skin of the affected part ; the history shows a recent origin, and there are no signs of disease of the subjacent viscera.

Solid tumours springing from the ribs or cartilages are also unattended by evidence of cardiac or pulmonary disease ; they are usually well defined and localised, and are as a rule readily distinguished, by means of palpation, from all other causes of bulging.

3. Fluid accumulations in the left pleural sac ; solid tumours of the left lung ; solid tumours in the anterior mediastinum.

Diagnosis.—Fluid accumulations in the left pleural sac, which give rise to prominence of the præcordia, also produce bulging of the whole¹ of the affected (left) side, and are attended by the characteristic symptoms and signs of left pleural effusion.

Solid tumours of the left lung, and new growths in the mediastinum, seldom give rise to marked prominence of the præcordia. When sufficiently large to produce bulging of this part of the chest wall, they are attended with characteristic symptoms and signs, which it would be out of place to enumerate here.

4. Fluid in the sac of the pericardium.

5. Increase in the size of the heart itself, especially those enlargements which are attended with marked hypertrophy of the organ.

The characteristic features of prominence of the præcordia, due to fluid in the sac of the pericardium and enlargement of the heart itself, which are the most common causes of local bulging, and which are of course the most

¹ A small encysted pleuritic effusion might produce local bulging of the præcordia ; but such a condition is infinitely rare. A more frequent condition, but one which seldom gives rise to difficulties in diagnosis, is an empyema which is pointing.

important, so far as our present studies are concerned, will be afterwards considered in detail. It is important to remember that the prominence which results from these causes is greater in young persons (in whom the chest walls are elastic) than in old people.

6. Forward displacement of the heart by an aneurismal or solid tumour situated behind it. This condition is extremely rare.

7. The presence of an aneurism of the first portion of the aortic arch. The bulging in such cases is almost invariably above the fourth rib, but occasionally the aneurism 'points' lower down, as in a case which I shall afterwards describe.

Flattening or retraction of the præcordial region.

This condition may be due to :

1. Congenital malformation.

Diagnosis.—In these cases the depression is seldom limited to the præcordial region. It is usually symmetrical, and involves the lower sternal region, together with more or less of the mammary and infra-mammary regions on each side.

2. Long continued external pressure. Depression of the lower end of the sternum is sometimes seen in shoemakers, and is produced by the pressure of the 'last' against this part of the chest.

Diagnosis.—The depression which occurs in shoemakers is more central than the depression which results from cardiac affections, and involves the right as well as the left infra-mammary region. The central position of the depression and the nature of the patient's employment, together with the absence of any history, symptoms, or signs of local disease, are the points to which attention must be directed in making the diagnosis.

3. Retraction of the left lung, a condition which may result from pleurisy, cirrhosis of the lung, etc.

Diagnosis.—In these cases the retraction or depression is not confined to the præcordial region, but affects the whole of the left side ; while the associated physical signs, and the previous history, usually afford distinctive evidence of the condition.

4. Pericarditis.—Here the depression is usually confined to the lower end of the sternum and the adjacent part of the left infra-mammary region. The depression does not occur until absorption of the inflammatory products, and adhesion of the opposed surfaces of the pericardium have taken place. It is always best marked in those cases in which the chest wall is elastic (*i.e.* in young subjects), and in which the exterior of the pericardium has become adherent to the chest wall as the result of inflammation outside the sac.

*The position, extent, and character of the Visible Impulse,
especially the position of the Apex Beat.*

The visible impulse as a whole, and the position and characters of the apex beat in particular, must be noted—but since it is usually necessary to confirm and supplement the information derived from inspection by means of palpation, I shall leave the description of the cardiac impulse in health and in disease until that method of investigation has been considered.

Epigastric Pulsation.

Should this condition be present, it will of course be observed at this stage of the examination (*i.e.* during the inspection of the præcordial region); but since it is necessary to employ palpation to define its exact characters, it will be more conveniently considered after that mode of examination has been described.

Pulsations over the Aorta and at the Root of the Neck.

In addition to the visible cardiac impulse and the epigastric pulsation just mentioned, the presence or absence of pulsation in other parts of the thorax and at the root of the neck should be noted at this stage of the examination. It will, however, be more convenient to describe these pulsations when I come to treat of the systematic examination of the aorta and superficial vessels.

The Condition of the Integument over the Præcordia.

The condition of the integument over the præcordial region sometimes affords useful information.

Cupping marks are suggestive of previous inflammation; and a history of cupping during a previous attack of rheumatic fever is very suggestive of an acute rheumatic inflammation of the heart (pericarditis, or endocarditis).

Præcordial Vascularity.—It is common to observe, especially after middle life, a line of minute vessels running across the chest in the neighbourhood of the præcordial region. The late Professor Laycock, who first directed attention to the condition, considered that it was suggestive of cardiac disease, and there can be no question but that the condition is more frequent in persons suffering from vascular lesions than in other people, but it occurs not unfrequently in perfectly healthy individuals, and is not therefore a sign of much importance.

Edema of the subcutaneous cellular tissue, when it is limited to the præcordial region, usually depends upon a local inflammation, and is then associated with tenderness on pressure, redness, and the like. It may however depend upon disease of the subjacent viscera. The condition with which it is most frequently associated is a malignant intra-thoracic growth. Edema of the chest wall and of the præcordial region is also of course seen as part and parcel of a general dropsy.

PALPATION OF THE PRÆCORDIA.

Palpation corroborates the information derived from inspection, and, in many cases, enables us to ascertain with exactitude:—

1. The position of the apex beat.
2. The character of the cardiac contractions, *i.e.* the force, rhythm, celerity, etc., of the cardiac impulse.

In some cases it also affords additional information, inasmuch as it reveals the presence of cardiac thrills, or friction fremitus, and enables us to elicit the presence or absence of pain and tenderness on pressure over the præcordial region.

Method of palpating the Chest in order to ascertain the exact position of the Apex Beat.

In order to ascertain the exact position of the apex beat by means of palpation, the patient should be laid in the recumbent position, and the fingers of the right hand placed lightly over the front of the chest below the left nipple. The position of maximum cardiac impulse, which usually corresponds to the position of the apex beat, should then be noted, and the interspace in which the apex pulsation is situated,¹ and the distance of the apex beat from the left border of the sternum ascertained.

It sometimes happens that the apex beat is not appreciable when the patient is in the recumbent position. In these cases (the nature of which I will presently describe), the position of the apex beat can usually be ascertained by so altering the position of the patient as to bring the heart more in contact with the wall of the chest. He may be made to sit up and to lean forward, for instance; or, he may be placed on his left side. If the latter method be adopted, due allowance must be made for the lateral displacement of the heart, which results from the alteration in position.²

¹ To ascertain the exact position of the apex, the interspaces must be carefully counted from above downwards; the forefinger of the right hand should first be made to define the position of the first interspace immediately below the clavicle (in many persons there is a well marked prominence of the sternum at the level of the second costal cartilages, which forms an easy guide to the second interspace); the middle finger of the same hand should then be inserted between the second and third ribs; the second interspace having thus been ascertained, the forefinger of the right hand should be placed in it, and the middle finger should then, but not till then, be slipped over the third rib into the third interspace; the fourth and succeeding interspaces are successively defined in the same manner, until that in which the apex beat is situated, is finally reached. The position of the apex with regard to the left nipple (in males); or, better, its exact distance from the left border of the sternum, must then be measured and noted.

Note.—In fat or very muscular persons it may be necessary to use some force in defining the position of the interspaces in the manner just described.

² According to Sibson, the apex beat may, in some cases, be displaced as much as two inches to the left, by turning the patient from the back on to his left side.

The position of the apex beat is of great practical importance, for it corresponds to the lowest point of the heart, and is in fact the means by which we are in the habit of determining the lower boundary of the organ.

The normal position of the apex beat.

In the adult healthy male the apex can usually be seen and felt pulsating (over an area about an inch square) between the fifth and sixth ribs, at a point two inches to the left of the sternum.¹ In well nourished healthy persons, the apex pulsation is the only pulsation which is visible over the præcordia during ordinary (tranquil) action of the heart.

In women with well developed mammæ, in fat or muscular persons, and when the heart is acting feebly the apex beat may be invisible. *Vice versâ*, in thin persons, or where the heart is excited or hypertrophied, the apex beat is unusually well marked.

The position of the apex beat varies somewhat in different healthy individuals, being sometimes situated a little higher, sometimes a little lower than the fifth interspace. These alterations depend for the most part, as Sibson has shown, upon the length of the arch of the aorta; and also upon the age of the individual, the form of the thorax, and the condition of the respiratory organs.

In children the apex beat is usually higher, and often more to the left; *vice versâ*, in old people, and especially in those in whom the aorta is atheromatous and elongated, the heart, and therefore the apex beat, are lower than the fifth interspace.

In strong robust persons, and in those who possess a broad and deep chest of the inspiratory type, the position of the heart and arteries, and of all their parts, is lower, while in those who are slender, and possess a narrow and flat chest of

¹ In describing the position of the apex beat, more particularly in females, the interspace in which it is situated, and its exact distance from the left border of the sternum should be stated. In males the apex beat is normally situated an inch within, and an inch or an inch and a half below the left nipple.

the expiratory type, the position of these parts is higher than in the average healthy individual.¹

During inspiration the apex beat (in its relationship to the chest wall) is lower than during expiration. This is partly due to the fact, that the heart descends with the descent of the diaphragm, and partly because the front wall of the chest is raised during inspiration.

Congenital displacements of the Heart.

The heart is occasionally placed in the right side of the chest, the apex beat being somewhere in the neighbourhood of the right nipple. When this condition occurs congenitally, there is usually complete transposition of the viscera, the heart being placed on the right side, and the liver, for example, on the left side of the body. Cases of this description are not very uncommon, and two examples have come under my own personal observation.

A much rarer form of congenital displacement is that in which the heart is placed on the right side, while the liver and other viscera are situated in their usual position. The following case of this description recently came under my notice; and another case—verified by *post-mortem* examination—has lately been recorded in the pages of the *Lancet*.²

Case I.—Congenital displacement of the heart to the right side, the position of the other viscera being normal.

J. A., æt. 39, a joiner, presented himself at the Cowgate Dispensary, Edinburgh, in December 1881, suffering from a dislocation of the shoulder. On stripping him, the house surgeon (Dr Christie) noticed strong pulsation just above the right nipple—while the impulse of the heart could not be felt in the usual position. Concluding that the case was one of congenital displacement Dr Christie kindly asked me to see the case; and with his opinion I entirely concurred.

The facts which I elicited were as follows:—The patient, who was somewhat thin and of bad *physique*, stated, that he had always enjoyed good

¹ Sibson *On the Position and Form of the Heart.* Russell Reynold's *System of Medicine*, vol. iv. p. 97.

² *Lancet*, July 8th, 1882, p. 9.

health; that he had never, to his knowledge, had anything the matter with his chest. He was not short of breath, had never suffered from palpitation or pain in the region of the heart, and had never had any symptoms or sensations which made him suspect that there was anything wrong with his heart.

The left infra-mammary region looked flattened, the depression being greatest in the sixth and seventh interspaces. The heart impulse could not be felt in the usual position. On percussion the left infra-mammary region was hyper-resonant; and the normal cardiac dulness could not be detected. The respiratory murmur was audible over the usual position of the heart, but the heart sounds could not be heard.

Very distinct pulsation could be seen and felt in the third, fourth, and fifth right interspaces (see fig. 25); there was marked dulness over the area of pulsation; and the heart sounds were loudly heard over the same part of the chest. The percussion note over the outer part of the right infra-clavicular region was impaired, but the right lung, both anteriorly and posteriorly, seemed normal. The liver was situated on the right side; and the stomach in its normal position—no splenic dulness was detectable. The girth of the right side of the chest was considerably larger than that of the left, the measurements being as follows:—

Position of Measurement	Right.	Left
At the level of the second rib	16½ in.	15½ in.
Through the nipples	15½ in.	13½ in.
At the level of the seventh rib	15 in.	14 in.

The heart's action was readily excited. Both sounds were accentuated, but free from murmur.

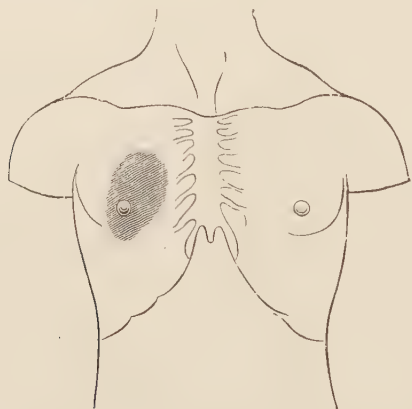


FIG. 25.—*Front view of the thorax in the case of J. A., showing the area of cardiac pulsation.*

Diagnosis.—The heart was obviously placed on the right side of the chest. The facts, that there was no history or evidence of lung disease, and that the girth of the right chest was considerably greater than the left seemed to show that the displacement was not due to retraction of the right lung, but that it was in all probability a congenital condition. The peculiarity of the case consisted in the fact, that the heart was the only organ transposed, the liver and stomach being placed in their normal positions.¹

Alterations in the position of the Heart and Apex Beat which occur in disease.

In studying the alterations in the position of the apex beat which occur in disease, it is important to remember that the organ rests upon the diaphragm, to which it is *indirectly* attached by means of the pericardium, and that it is *directly* attached at its base to the great vessels, and behind to the roots of the lungs by the pulmonary veins.

The organ therefore ascends and descends with the ascent and descent of the diaphragm ; further, it is readily displaced from side to side by lateral pressure.

The alterations in the position of the heart and apex beat which result from disease, may be due either to *extrinsic* or *intrinsic* causes.

A. Displacement of the Heart due to extrinsic causes.

The heart may be *pushed* to one side by fluid, gaseous, or solid accumulations or by enlargements of the surrounding viscera ; or, it may be *pulled*² to one side by retraction of the air containing viscera, *i.e.* the lungs ; or, again, it may, by the force of gravity, fall with the descent of the diaphragm.³

¹ Breschet and Otto, quoted by Peacock, in his work on *Malformations of the Human Heart*, page 2, have met with cases of this description ; but so far as I have been able to ascertain, no other cases have been recorded in English medical literature.

² The contraction of the lung tends to produce a vacuum within the thorax, in consequence of which the adjacent organs, including the heart, are of course displaced (pushed rather than pulled) towards the affected side by the atmospheric pressure.

³ When the diaphragm descends, the great vessels (and therefore the heart) are also dragged down by reason of their attachment to the pericardium.

The displacement may be either to the right or to the left, upwards or downwards, forwards or backwards.

Displacement of the Heart and Apex Beat to the right may be due to:—

1. The presence of fluid, gaseous, and solid accumulations in the left pleural sac, or solid enlargements of the left lung.

2. Retraction of the right lung—a condition which usually results from pleurisy with effusion, cirrhosis or phthisis.

Fluid in the left pleural sac (see fig. 26) is by far the most common cause of displacement to the right; and in

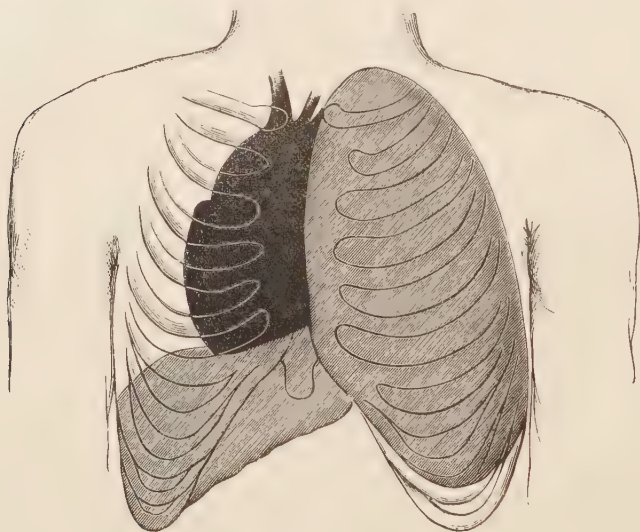


FIG. 26.—*Displacement of the heart to the right as the result of effusion into the left pleural cavity. (Modified from Sibson.)*

these cases the apex beat not unfrequently corresponds to the right nipple. Extreme displacement to the right may also be due to retraction of the right lung. Sibson quotes several cases of this description,¹ and I have seen more than one case in which the pulsation of the heart was situated just above the right nipple.

¹ Russell Reynold's *System of Medicine*, vol. iv. p. 143.

Displacement of the heart and apex beat to the left may be due to :

1. Accumulation of fluid, gaseous, or solid matter in the right pleural cavity ; or solid enlargements of the right lung or right lobe of the liver.
2. Retraction of the left lung (see fig. 27).

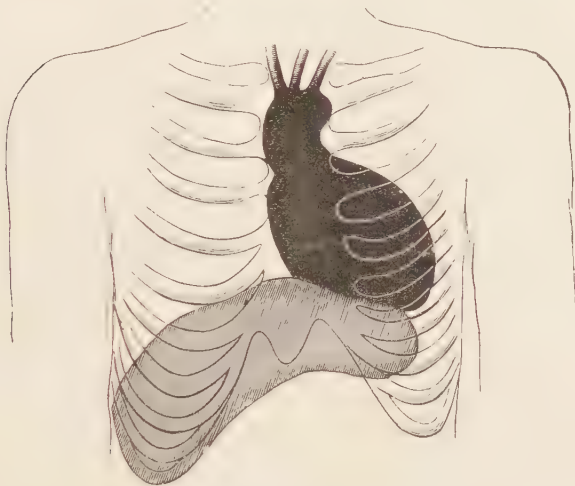


FIG. 27.—*Displacement of the heart to the left, in consequence of retraction (cirrhosis) of the left lung. (Modified from Sibson.)*

The displacement, which results from this cause is seldom so great as in the following case, in which the pulsations of the heart were observed in the left axilla.

Case II.—Pleurisy : Cirrhosis (?) of the left Lung ; great displacement of the heart to the left.

J. M., æt. 21, a fireman, was admitted to the Newcastle-on-Tyne Infirmary, under my care, on 3d December 1874, complaining of palpitation, shortness of breath, cough, spit, and great debility.

Previous history.—The patient, who has always been very round shouldered, stated that he had enjoyed excellent health until four years ago, when he had an attack of rheumatic fever, which laid him up for fourteen weeks : he does not know whether his heart was affected or not. After this attack he enjoyed good health for some years, when he ‘caught a severe cold,’ in consequence of exposure to cold and wet, and was ill for several weeks with cough and severe pain in the left side, which ‘caught

his breath.' These symptoms gradually subsided, and he continued in excellent health until four months ago, when his present illness commenced with cough, spit, and shortness of breath. These symptoms have gradually increased, and he has lost a great deal of flesh.

Present condition.—He is considerably emaciated. The girth of the left chest is considerably less than that of the right, the measurements being as follows :—

Position of measurement.	Right.	Left
At the second rib	15 ½ in.	14 ¾ in.
At the nipples	15 ½ in.	14 ½ in.
At the ensiform cartilage	15 in.	15 in.

The percussion note over the greater part of the left lung, but especially at the base, is impaired; increased vocal resonance, sibilant râles, and occasional large crepitations are heard on auscultation. There was also evidence of commencing consolidation of the right apex. The pulsation of the heart can be very distinctly seen and felt in the left axillary and infra-axillary regions, the apex being apparently situated in the sixth interspace, two inches outside the nipple. The percussion note between the left nipple and the sternum is resonant, and the heart sounds are inaudible at this spot; at the aortic cartilage the heart sounds are very faint, but are much louder in the second and third interspaces on the left side.

Upward displacement may be due to :

(1) Anything which raises the arch of the diaphragm, such as solid, liquid, or gaseous accumulations in the cavity of the abdomen—ascites (see fig. 28), ovarian tumours, tympanites, enlargement of the liver (see fig. 29), fibroid tumours of the uterus, etc.

In many of these cases the displacement is partly to one side as well as upwards. Enlargement of the left lobe of the liver, for example, usually displaces the heart upwards and to the left, while great enlargement of the spleen may push it upwards and to the right.

(2) Retraction of the upper part of either lung, the most common pathological condition being phthisis.

Downward displacement may be caused by :

(1) Anything which presses down the heart or diaphragm, such as aneurisms or solid tumours, emphysema of the lungs, etc. (see fig. 30).

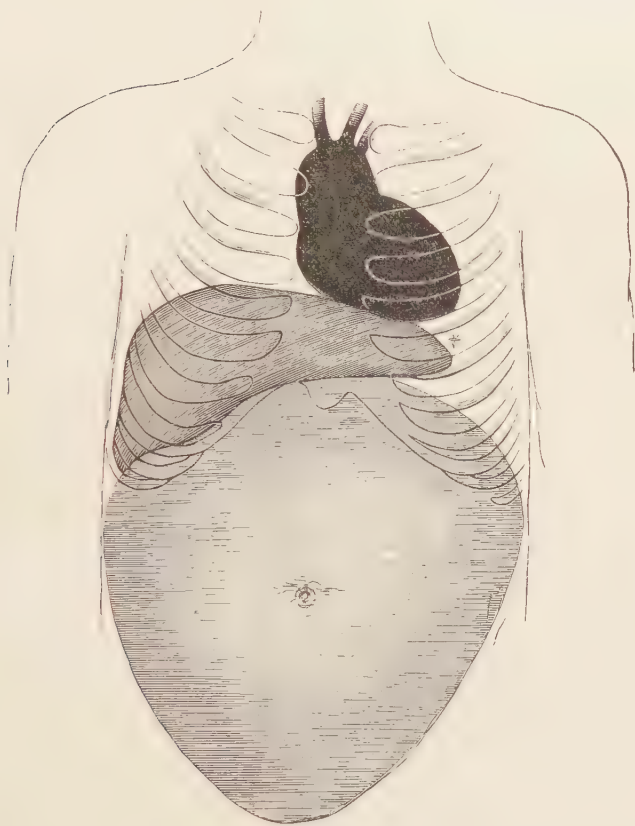


FIG. 28.—*Upward displacement of the heart, the result of ascites.*

(2) Collapse of the abdominal viscera, and consequent descent of the diaphragm.

Forward displacement is very rare, but it does sometimes result from an aneurism (as in a case which I shall afterwards relate) or a solid tumour in the posterior mediastinum.

Backward displacement is still more rare; it is said to be occasionally produced by tumours or inflammatory accumulations in the anterior mediastinum, but no case of this description has come under my personal observation.

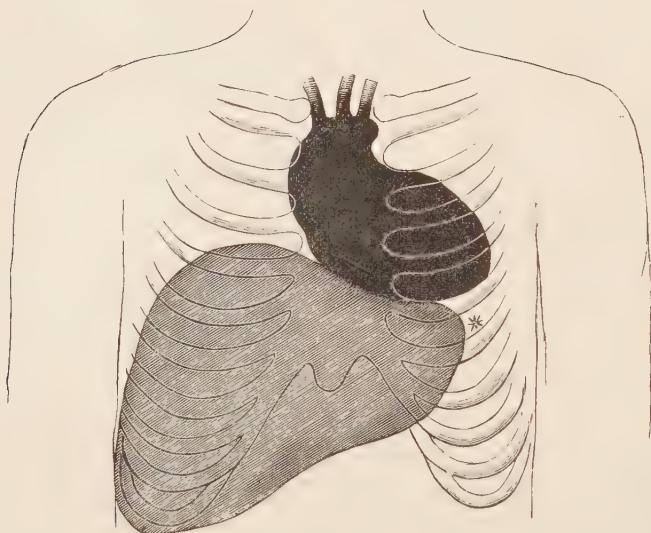


FIG. 29.—Displacement of the heart upwards and to the left, the result of enlargement of the liver. (Modified from Sibson.)

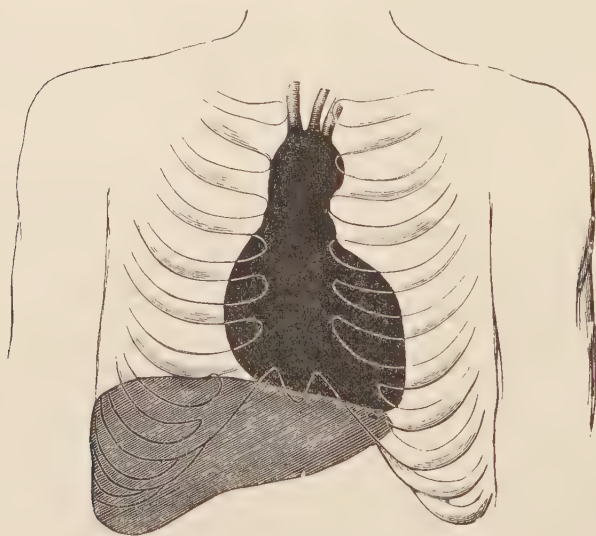


FIG. 30.—Downward displacement of the heart, the result of pulmonary emphysema. (After Sibson, modified.)

B. Displacements of the apex beat due to intrinsic causes.

The intrinsic conditions which may produce displacement of the heart and apex beat are —

1. *Fluid in the sac of the Pericardium.*—Where the pericardial effusion is considerable, but not excessive, the base of the heart and the great vessels are pushed upwards and backwards, and the apex is tilted upwards and outwards, so that it may correspond in position to the left nipple, or in some cases may be situated still further to the left.

Where the effusion is very copious, the apex beat may be completely obscured; while in those cases in which the amount of effusion is small (the heart itself being of normal size), or in which the heart is fixed by old pericardial adhesions, there is little or no alteration of the apex beat.

2. *Alterations in the shape and size of the heart itself.*—When the left ventricle is hypertrophied (see fig. 31), the extension is chiefly downwards and outwards (*i.e.* to the left), and the apex beat may be situated in the sixth, seventh, or even the eighth interspace, three, four, or even five inches to the left of the sternum. In many cases it is considerably outside the left nipple.

In hypertrophy and dilatation of the right ventricle, the apex beat (which is usually under such circumstances more diffused than in health), is displaced downwards and to the right (see fig. 32), and the cardiac impulse is not unfrequently best seen and felt in the pit of the epigastrium. But since epigastric pulsation may be due to several other important conditions, it will perhaps be well to enumerate them here.

Epigastric Pulsation.

The chief forms of epigastric pulsation are, in the order of their relative frequency, as follows:

(1.) *Transmitted pulsation from the heart.*—This form of pulsation is generally due to enlargement (hypertrophy and dilatation) of the right ventricle, but it may also be caused by the downward displacement of the organ, which is seen in pulmonary emphysema,¹ aneurism of the arch of the aorta,

¹ In emphysema the right side of the heart is dilated and hypertrophied, and to this cause the epigastric pulsation, which is so marked a feature in emphysema, is in part due.

collapse of the abdominal viscera, etc. The pulsation is in these cases exactly synchronous with the ventricular contraction, and is often transmitted through the left lobe of the liver.

(2.) *Pulsation of the abdominal aorta.*—In some of these cases, as for instance in neurotic pulsation of the vessel, the aortic pulsation may be *directly* felt, in others it is *indirectly* felt, and is communicated through the liver or some solid body, such as a cancer of the pylorus, lying in front of the

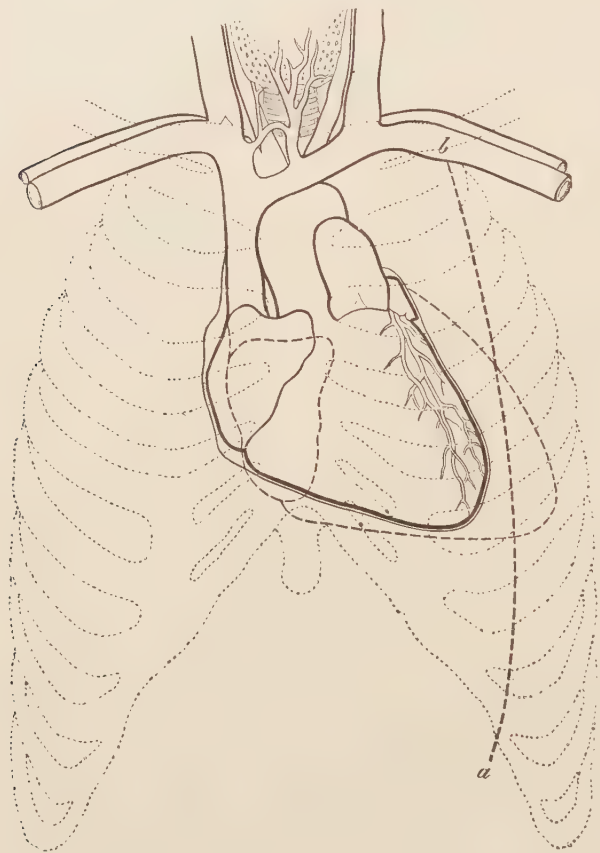


FIG. 31.—*Hypertrophy of the left ventricle, showing the altered position of the apex.*
(After von Dusch.)

The continuous line represents the normal heart; the dotted line the hypertrophied left ventricle. The apex of the left ventricle is outside the dotted line a b, which is drawn through the left nipple.

vessel. The pulsation in this form is a little behind (after) the apex beat, but this fact is sometimes with difficulty established in practice.

(3.) *Aneurism of the lower part of the thoracic, or of the upper part of the abdominal aorta.*—This is not a common cause of epigastric pulsation. The aneurismal pulsation may be directly felt, or may be transmitted through the liver.

According to Sibson, the epigastric pulsation, which is caused by an aneurism of the lower part of the thoracic or

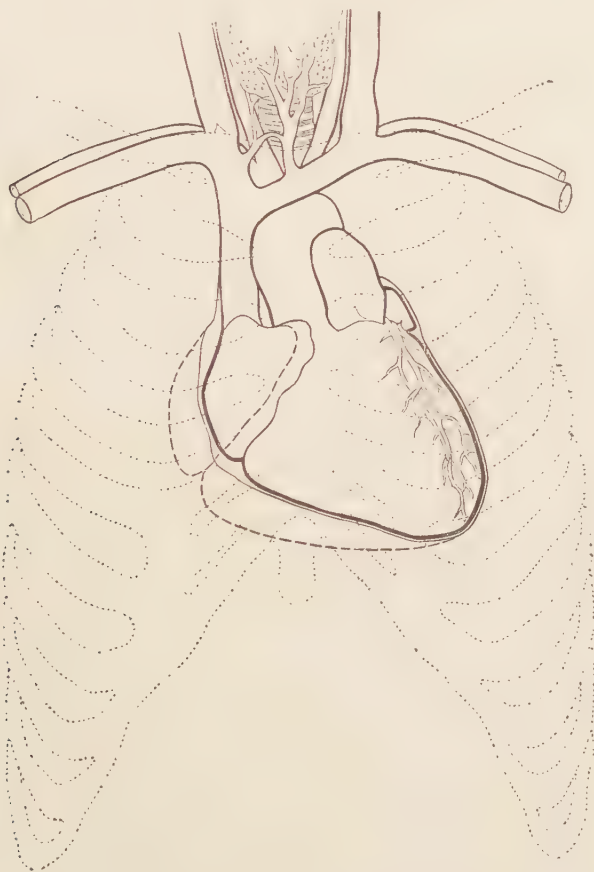


FIG. 32.—*Hypertrophy of the right ventricle.* (After von Dusch.)

The normal outline of the heart is represented by a continuous line: the hypertrophied right heart by a dotted line.

the upper part of the abdominal aorta, is strong during expiration, but is lessened, or even disappears, during inspiration. In cases of abdominal collapse, on the contrary, the epigastric pulsation is more marked when the patient takes a deep inspiration.¹

(4.) *True Pulsation in the liver.*—Here the pulsation is caused by a 'back-wash' from the right auricle. The liver is always enlarged, and the pulsation is diffused through, and not simply communicated to it. Its rhythm corresponds to the apex beat, for the blood-wave which produces it is propelled by the right ventricle through the incompetent tricuspid orifice into the hepatic veins. This form of pulsation is therefore pathognomonic of tricuspid regurgitation. In some cases of this description the hepatic pulsation may possibly be due to communicated pulsation from the inferior vena cava,² for the back-wash also extends into that vessel.

THE EXTENT OF THE CARDIAC IMPULSE AS ASCERTAINED BY INSPECTION AND PALPATION.

In health, and during tranquil action of the heart, the apex beat is the only cardiac pulsation which is visible, and it usually covers an area of about one square inch in extent. When the heart is excited, in thin persons, and when an unusually large portion of the organ is in direct contact with the chest wall—a condition which may result from increased size of the organ, forward displacement of the heart, or retraction of the anterior margins of the lung—pulsation may be seen and felt over a large portion of the præcordial region. Diffuse wave-like pulsation is seen in some cases of dilatation of the organ, and, according to some observers³ in pericardial effusion.

¹ Russell Reynold's *System of Medicine*, vol. iv. p. 129.

² Pulsation in the vena cava might also result from a communication between a large arterial trunk, such as the abdominal aorta, and that vessel; but I am not acquainted with any case in which this condition has been observed.

³ It is very doubtful whether a diffuse wave-like pulsation over the præcordial region does result from pericardial effusion.

The exact character of the Cardiac Impulse with respect to force, rhythm, celerity, etc.

The force of the cardiac impulse is ascertained by palpating the præcordia in the manner described above, and is still more accurately observed by placing the right hand over the præcordial region, the left hand over the back between the scapulæ, and by compressing the chest firmly between the two. The pulsation of deep-seated aneurisms can sometimes be observed by this method, when the ordinary method of palpation fails to elicit any abnormal pulsation.

The force of the cardiac impulse varies considerably in different cases, and depends upon:—(1) The thickness and character of the media (tissues of the chest wall, lung, etc.) which separate the heart from the hand of the observer; (2) The force with which the heart is contracting.

Diminution or absence of the cardiac impulse (as seen and felt over the præcordial region) on the one hand, and excessive impulse on the other, do not, therefore, *necessarily* indicate *actual* diminution or *actual* increase of the cardiac contractions,—though they certainly suggest these conditions.

Diminished Cardiac Impulse.

We will first consider those cases in which the cardiac impulse, as seen by the eye and felt by the hand, is weakened or abolished. In these cases, as I have just stated, the force of the cardiac contraction is sometimes apparently, sometimes actually, weakened.

The conditions which give rise to diminution or absence of the cardiac impulse, the force of the cardiac contractions being normal (i.e. to apparent diminution of the force of the cardiac contractions), are as follows:—

1. Undue thickness of the thoracic walls, especially the presence of a thick layer of subcutaneous fat, largely developed mammæ, etc.¹

¹ In these cases (where the subcutaneous fat is excessive) the heart may be infiltrated with fat, or in a condition of true fatty degeneration, and the force of the cardiac contractions may be *actually* as well as *apparently* diminished.

2. Excessive over-lapping of the heart by the lungs. This condition is seen even in health where the thorax is unusually short and broad, and the lungs voluminous ; but it reaches its highest degree of development in pulmonary emphysema.

3. Fluid in the sac of the pericardium. When the effusion is scanty, the position and force of the apex beat may not be altered. When the effusion is more considerable, the apex is raised and carried more to the left, and the cardiac impulse as felt by the hand is weakened.¹ When the effusion is very great, the apex beat may completely disappear,² and it may be impossible, when the patient is lying on his back,³ to feel any cardiac impulse.

The conditions which give rise to actual diminution of the force of the cardiac contractions, and therefore of the cardiac impulse, as felt by the hand externally, are :—

1. General collapse, and anything which temporarily weakens the action of the heart—(nervous depression, excessive smoking, etc).

2. Structural alterations which weaken the cardiac muscle, such as fatty degeneration, fibroid degeneration, acute myocarditis, etc.

Increased Cardiac Impulse.

Increased cardiac impulse, as seen and felt over the præcordia, like diminished impulse, may be either *apparent* or *real*.

The conditions which gives rise to increased cardiac impulse—the force of the cardiac contractions remaining normal—(apparent increase) are as follows :—

1. An atrophied condition of the chest walls. This cause

¹ In some of these cases the visible impulse may be considerable, but the impulse, as felt by the hand, slight.

² In many cases of excessive pericardial effusion, the force of the cardiac contractions is also *actually* impaired.

³ The cardiac impulse can generally be felt if the patient is made to lean well forwards, or turn on to his left side.

is, to a large extent, theoretical and of little importance in practice. Atrophy of the subcutaneous fat, and of the chest muscles (the lungs being normal), probably exerts little influence on the cardiac impulse as felt externally. Indeed in many of these cases the heart is itself debilitated, and as a matter of fact the impulse weakened.

2. Increased exposure of the heart. This is a frequent cause of increased pulsation, and is usually due to the retraction of the lungs, which results from pleurisy, cirrhosis, etc.; but it may be due to forward displacement of the organ.

The conditions which produce actual increase of the force of the cardiac contractions, and therefore of the cardiac impulse, as seen and felt externally, are :—

1. All conditions which produce temporary over-action of the heart, such as violent exertion, mental agitation, and the numerous causes of functional palpitation.

2. Hypertrophy of the heart; particularly hypertrophy of the left ventricle. In these cases the cardiac impulse is heaving or pushing, and the whole chest may appear to be raised *en masse*.

The Rhythm of the Cardiac Contractions.

When the cardiac impulse is perceptible, the exact rhythm of the cardiac contractions can of course be ascertained by palpating the præcordia, but the rhythm of the heart will be more appropriately considered when I come to treat of the pulse.

The Celerity of the Cardiac Impulse.

In some conditions, the heart contracts more abruptly than in health, and the impulse, as seen and felt externally, is correspondingly sudden. This form of impulse (due to increased celerity of contraction) is chiefly seen in conditions of nervous excitement, and of 'irritable weakness,' such as is associated with anæmia and allied conditions. The area of visible impulse is often increased.

In other cases the cardiac contraction, and therefore the

impulse as seen and felt externally, are unusually slow and laboured (diminished celerity of impulse). This form of contraction is generally associated with hypertrophy, more especially hypertrophy of the left ventricle; the cardiac impulse is usually strong and pushing, and in many cases the chest appears to be raised up *en masse*.

Præcordial thrills and friction fremitus.

Vibratile sensations, of a 'soft' trembling character, and resembling very closely the vibratile sensation which is experienced on placing the hand over the back of a cat, when purring (hence the term 'purring tremor' which has been applied to them), are sometimes felt on placing the hand over the præcordia. To vibrations of this description the term *thrill* is applied. The term *friction fremitus* is given to vibrations of a harsher, rougher, and grating character, which can be felt when the hand is placed over the chest.

Thrills may be produced either in the heart itself or in the great vessels. The cardiac thrills with which we are now concerned, are usually produced by the passage of the blood stream through a stenosed or roughened orifice, and are therefore generally produced by a 'direct' current.

Stenosis of the mitral valve is the most common cause of cardiac thrill. The vibratile sensation is felt in the mitral area, *i.e.* over the position of the apex beat; its rhythm is (for the reasons which I shall afterwards explain in speaking of cardiac murmurs) *presystolic*, that is to say, it occurs immediately before the contraction of the ventricle, *i.e.* immediately before the apex beat. Aortic stenosis is the next most common cause of thrill. The vibratile sensation is *systolic* in rhythm, and is felt over the base of the heart and ascending thoracic aorta.

Occasionally, however, the thrill is produced by a backward or regurgitant current. A systolic thrill over the apex is felt in some cases of mitral regurgitation; and a diastolic thrill at the base is not at all uncommon in aortic regurgitation. Thrills produced at the tricuspid and pulmonary orifices are extremely rare.

Friction fremitus is produced by the rubbing together of two rough surfaces. When felt over the præcordia, it may be pericardial or pleural.

Pericardial friction fremitus corresponds in rhythm to the rhythm of the heart. It is a comparatively rare phenomenon which is seen in some cases of pericarditis, more especially in those cases in which the lymph coating of the pericardial sac is tough. Pericardial friction fremitus is therefore observed towards the later periods of pericardial inflammation, *i.e.* when the lymph is becoming tough and organised. The clinical significance of pericardial fremitus is not great, for friction vibrations are almost always more apparent to the ear than to the touch, and are therefore better studied by auscultation than by palpation.

Pleural friction is sometimes felt over the præcordial region, more especially over the borders of the heart. Its rhythm corresponds to the rhythm of the respirations, and by this means it is distinguished from the friction fremitus of pericarditis.

PERCUSSION.

Percussion, the next method of physical examination which we employ, is an extremely important means of investigation. It is founded on the facts:—(1) that the heart is a solid organ which is in great part surrounded by the air-containing lungs; and (2) that the 'sound' and 'sensation of resistance,' which are obtained by percussing a solid body and an air-containing viscus, are markedly different.

The object of percussion is to ascertain the size, the shape, and position of the heart; and in order to determine these points we must endeavour to define:—(1) the extent and outline of that part of the heart which is uncovered by lung,—*the area of superficial or absolute cardiac dulness*; (2) the size and outline of the heart as a whole—*the area of deep or relative dulness*, as it is sometimes termed—(the area of *impaired* percussion).

The area of superficial or absolute cardiac dulness.

The size of the area of superficial or absolute cardiac dulness varies considerably, in health, with the condition of the lungs, being diminished during inspiration, and increased during expiration, and is subject to marked alterations in disease.

Theoretically, it should exactly correspond to that portion of the heart which is uncovered by lung (see fig. 33), but as a matter of fact its inner (central) border does not correspond to the mid-line of the sternum, but to the left border of that bone;¹ while its inferior border cannot be accurately determined by means of percussion.²

The extent of the heart, which is uncovered by lung, during moderate inspiration, is seen in figure 33. In full inspiration, the anterior border of the right lung closely corresponds to a line drawn vertically downwards through the centre of the sternum. It extends downwards as far as the junction of the sixth costal cartilage with the sternum; the lower border of the lung leaves the middle line usually at the level of the sixth rib or sixth interspace, and passes obliquely downwards and backwards.

The anterior border of the left lung closely corresponds to the mid-line of the sternum until it reaches the level of the fourth costal cartilage. At this point it passes obliquely downwards and outwards until it reaches a point which usually corresponds to the junction of the fifth rib with its costal cartilage. It then turns obliquely inwards and downwards, forms the tongue-shaped projection, which is well shown in the figure, and finally passes backwards and downwards, usually leaving the middle line at the level of the sixth rib or sixth inter-space.

The area of superficial or absolute cardiac dulness is, then, more or less triangular³ in shape; the size of the triangle varying with the position of the anterior borders of the lungs.

¹ The sternum is an excellent conductor of sound, and it is difficult or impossible when one lateral half is in contact with the resonant lung, as it is during complete inspiration, to get an absolutely dull percussion sound over the other (the right) half of the bone.

² The lower border of the heart cannot be accurately defined by percussion, for it is practically impossible to distinguish the dull sound which is due to the lower part of the heart, and the dull sound which is due to the upper part of the liver.

³ In many cases, the area of superficial cardiac dulness is rather quadrangular than triangular.

The *base* of the triangle cannot, for the reason already given (see foot-note, page 122), be accurately defined by percussion, but is represented with sufficient accuracy by a horizontal line drawn from the position of the apex beat to the mid-sternum. Under normal circumstances, and during moderate inspiration, the length of the base line is about $2\frac{1}{2}$ inches.

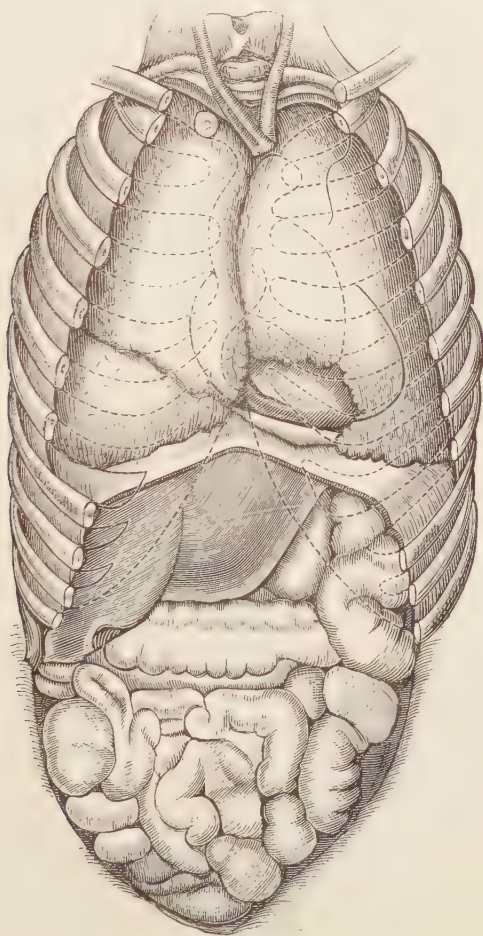


FIG. 33.—*Superficial view of the organs of the chest and abdomen from the front, showing the part of the heart uncovered by lung. (Enlarged from Sibson.)*

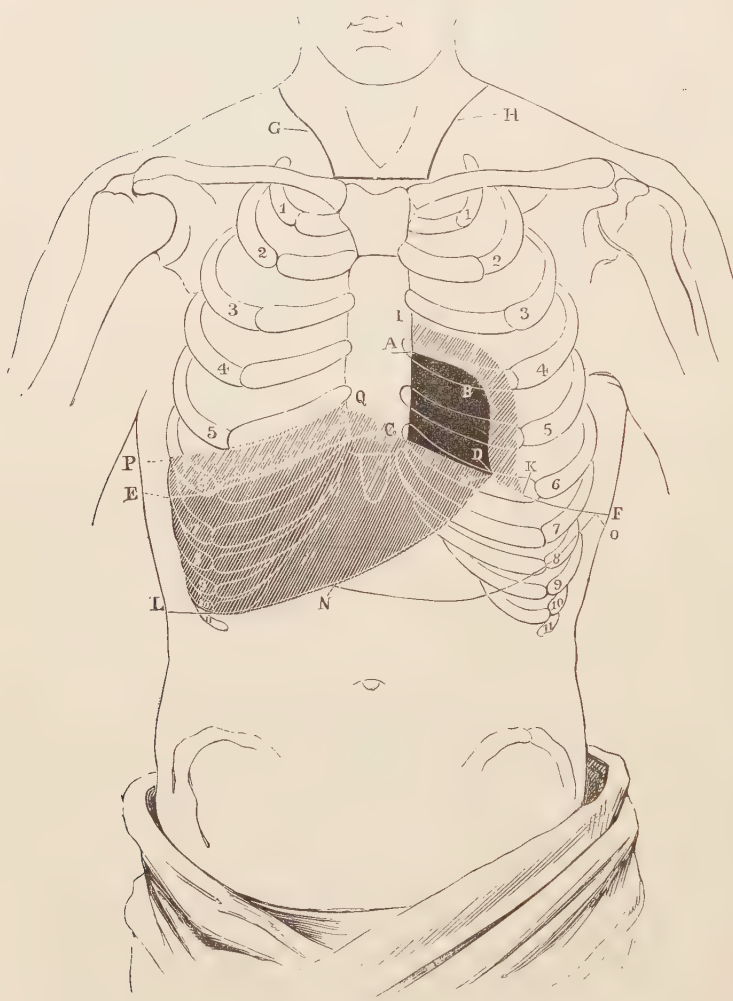


FIG. 34.—*The area of præcordial dulness in middle age.* (After Weil.)

ABCD, area of superficial or absolute cardiac dulness; AEK, area of impaired percussion or deep dulness; CE, lower border of right lung; DF, lower border of left lung; G and H, upper borders of lungs; PQ, upper border of hepatic dulness; LM, lower border of hepatic dulness; NO, lower border of stomach in moderate distention.

The *apex* of the triangle corresponds under normal circumstances to the junction of the fourth left costal cartilage with the sternum.

The *vertical side* of the triangle, *i.e.* the side formed by a line drawn from the apex of the triangle to the central (the sternal) end of the base line, is, under normal circumstances, about two inches in length. For the reason already given (see foot-note, page 122) it does not correspond to the mid-line of the sternum, but to the left border of the bone.

The third or *oblique side* of the triangle, which is seldom a straight line, but generally more or less curved, as represented in figures 34 and 35, is formed by drawing a line from the apex of the triangle to the outer end of the base-line (*i.e.*, to the apex of the heart); it is usually about 3 inches in length.

Mode of percussing the area of superficial or absolute cardiac dulness. In seeking to determine the exact size and boundaries of the area of superficial or absolute cardiac dulness, at the bedside, it is convenient in the first place, as recommended by Sansom, to draw the base-line, *i.e.* to draw a horizontal line from the position of the apex beat to the mid-sternum, and then to determine the position of the vertical and oblique sides of the triangle respectively, *i.e.* to define the anterior margins of the lungs by means of percussion. And it is important to remember that at those points at which the absolute and relative areas of cardiac dulness meet (*i.e.* where the margins of the lungs terminate), the percussion strokes must be lightly struck, for otherwise the resonant note which is obtained from the thin layer of lung will be, to some extent, obscured by the dull sound which is obtained from the solid heart, which is situated beneath it. The extent of the area of absolute cardiac dulness, both in moderate inspiration, and during complete inspiration, and in complete expiration, should be ascertained if extreme accuracy is required, percussion being performed, while the patient holds his breath, after a moderate inspiration, a complete inspiration, and a complete expiration, respectively.

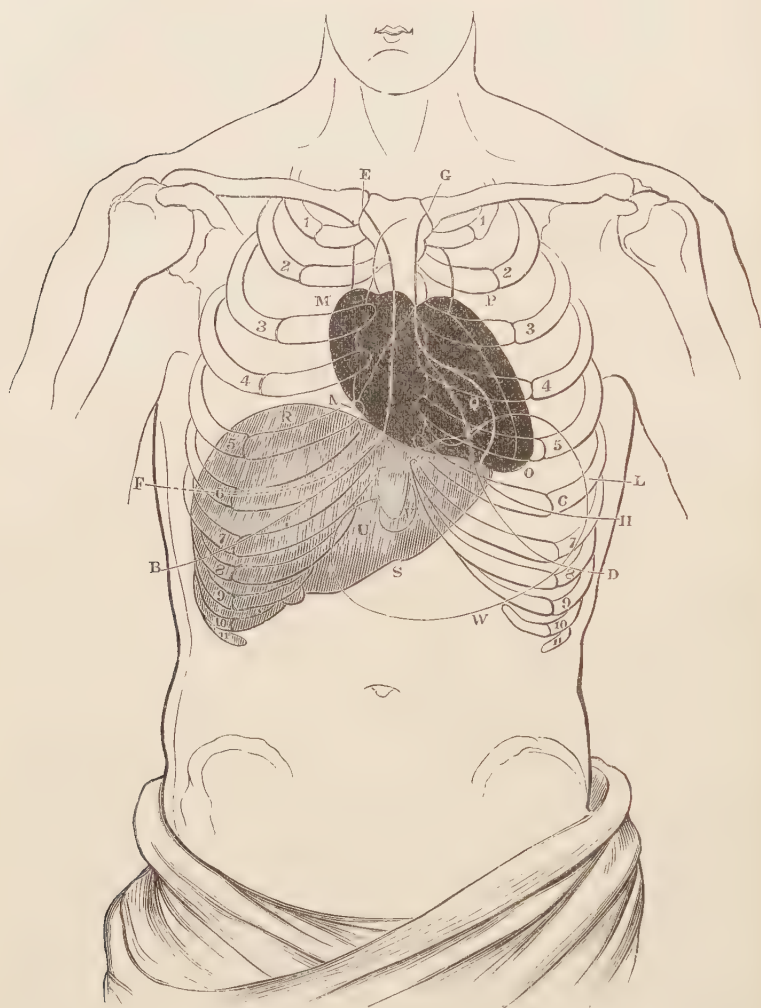


FIG. 35.—*Anterior view of the chest and abdomen, showing the position of the heart in the thorax and its relationship to the surrounding viscera.* (After Weil.)

EF, edge of the right lung; GH, edge of the left lung; MN, right border of the heart; NO, lower border of the heart; PO, left border of the heart; Q, sinus mediastinocostalis, situated between the edge of the pleura and incisura cardiaca of the anterior border of the left lung; R, highest point of the portion of liver covered by lung; S, lower edge of the liver; T, cardiac portion of the stomach; U, pyloric portion of the stomach; V, small curvature of the stomach; W, greater curvature of the stomach.

The area of deep or relative cardiac dulness.

The area of deep or relative cardiac dulness should correspond to the space which the heart occupies as a whole, that is to say, it should extend vertically from the third rib or second interspace to the base-line drawn from the apex beat horizontally across the sternum, and transversely from an inch to the right of the sternum to a point slightly within the left nipple. (See fig. 35.) As a matter of fact, however, it is difficult to define the exact extent of the deep cardiac dulness. As we have already seen, its lower border cannot be accurately ascertained by means of percussion. Its upper boundary runs into the dulness which is due to the presence of the great vessels (aorta, pulmonary artery, and superior cava, etc.); while the right and left borders of the heart (more particularly the right) are so deeply situated, that their exact position is with difficulty determined.

The left border can generally be fairly well defined,—and we have an additional guide to its position in those cases in which the apex beat is well-marked, for the position of the left apex beat not only represents the lowest, but also the outermost point of the heart. (In some cases of pericardial effusion, and in some cases of hypertrophy of the left ventricle, the cardiac dulness extends farther to the left than the position of the apex beat.) When the right ventricle is dilated, the left apex beat may be ill defined or absent. In cases of this description the pulsation of the right ventricle is often best marked in the epigastrium, and the position of the apex beat affords no guide to the left border of the heart.

Mode of percussing the deep cardiac dulness.—The observer should first endeavour to define the upper border of the heart by percussing from above downwards in the para-sternal line.¹ He should then determine the extent of the transverse dulness at the level of the fourth rib or fourth interspace. The percussion must be forcibly made, for the borders of the heart are covered by a thick layer of resonant lung; small differences in tone must be accurately noted, and the ‘percussion resistance’ carefully observed.

¹ The para-sternal line is an imaginary line drawn vertically downwards over the front of the chest mid-way between the left border of the sternum and the left nipple.

Alterations in the extent of the cardiac dulness which occur in disease.

The area of cardiac dulness may be either increased or diminished in disease; and both conditions—increased and diminished dulness over the præcordia—may be either *actually* or *apparently* due to cardiac alterations. In other words, alterations in the extent of the præcordial dulness are in some cases due to alterations in the size of the heart and pericardium; in other cases, to alterations in the surrounding viscera (more particularly in those portions of the lungs which overlap the heart), the size of the heart and pericardium being normal.

Increased dulness over the præcordia.

Increased dulness over the præcordia may be due to:—

1. Increase in the size of the heart (hypertrophy, dilatation, deposits of fat on the exterior of the heart, new growths in its substances).

2. Fluid in the sac of the pericardium (pericarditis with effusion, hydro-pericardium).

3. Increased exposure of the heart—the organ itself being of normal size. Under this head are included:—

(a) Those cases in which the anterior borders of the lungs are retracted from pleuritic adhesions, cirrhosis, etc.

(b) Those rare cases in which the heart is displaced forwards by aneurisms or solid tumours in the posterior mediastinum.

4. Solid or fluid accumulations in contact with the heart, amongst which the following are the most important:—

(a) Consolidations (from pulmonary apoplexy, pneumonia, tubercle, new growths, etc.), of those portions of the lungs which overlap the heart.

(b) Fluid in the pleural sacs.

(c) Solid tumours, deposits of fat, or inflammatory formations in the anterior mediastinum.

(d) Enlargements of the liver.

(e) Aneurisms springing from the base of the aorta.

Note.—It is extremely important to remember that the dulness which, under ordinary circumstances, results from enlargement of the heart and

many of the other conditions which I have enumerated in the text, may not be present in those cases :—in which the lungs are emphysematous ; in which the anterior margins of the lungs are fixed by adhesions ; or in which there is air in the pericardial or pleural sacs.

The differential diagnosis of increased dulness over the præcordia.

Given the presence of increased dulness over the præcordia, we must, of course, endeavour to determine the exact nature of the pathological condition which is present ; and in order to arrive at this conclusion, it is convenient to proceed by the following stages or steps :—

Step No. 1.—Is the increased dulness derived from the heart itself, or is it due to the presence of some non-resonant substance in contact with the organ ?

Step No. 2.—If the dulness is due to the presence of a non-resonant substance in contact with the heart, what is the exact pathological condition which is present ?

Step No. 3.—If the dulness is directly derived from the heart itself, does it result from increased exposure (apparent enlargement) of the organ, or from an actual increase in the size of the heart, including the pericardium ?

Step No. 4.—If the dulness is due to an actual increase in the size of the organ, does it result from the presence of fluid in the sac of the pericardium, or from an enlargement of the heart itself ?

Step No. 5.—If the dulness is due to enlargement of the heart itself, is the enlargement general or partial, and what is the pathological character and cause of the condition ?

Step No. 1.—*Is the increased dulness over the præcordia derived from the heart itself, or is it due to the presence of some non-resonant substance in contact with the organ ?*

As a rule there is little difficulty in coming to a correct conclusion on this point. As we have previously seen, the chief pathological conditions which give rise to dulness in the neighbourhood of the heart, are :—

(a) Consolidations of the adjacent portions of the lungs (apoplectic, pneumonic, tubercular, sarcomatous, etc.).

- (b) Fluid in the pleural cavity.
- (c) Enlargement of the liver.
- (d) Tumours, or inflammatory deposits in the anterior mediastinum.
- (e) Aneurism of the first portion of the aortic arch."

Now in most of these conditions—in the vast majority of cases met with in practice—the increased dulness is not confined to the limits of the præcordia, but extends often for a considerable distance into the surrounding regions of the chest.¹ In many cases too, it does not conform to the shape of the dulness which results from an enlargement of the heart or pericardium.²

There are, too, as a rule, other symptoms and physical signs indicative of the cause of the dulness. In consolidations of the lung, for example, cough, expectoration, and alterations of the respiratory murmur over the seat of the dulness (tubular breathing, râles, etc.), would probably be present.

In addition to these positive facts the negative evidence—that there are no signs nor symptoms of disease of the heart or pericardium—confirms the diagnosis.

The points, then, to which attention should be directed, in order to come to a conclusion as to the first step in the diagnosis are:—

1. The extent and outline of the dulness.
2. The presence of symptoms or physical signs indicative of disease of the adjacent parts.
3. The condition of the heart and pericardium, as determined by other methods of investigation.

Step No. 2.—If the dulness is due to the presence of a non-

¹ Dulness, resulting from a limited consolidation of those portions of the lungs in contact with the heart, might of course be limited to the præcordia. In such a case the other physical signs and symptoms (negative and positive) would be quite sufficient to determine the diagnosis.

² It would be extremely difficult, indeed in many cases impossible, to distinguish the dulness due to a small tumour or inflammatory accumulation in the anterior mediastinum, from the dulness which results from enlargement of the heart or pericardium. Fortunately limited tumours of this description are rare, and the difficulty in diagnosis is therefore seldom met with in practice.

resonant substance in contact with the heart, what is the exact pathological condition which is present?

This point can only be determined by a careful and accurate survey of all the 'facts of the case' (symptoms, physical signs, etc.); and it would obviously be out of place to attempt to detail here the numerous symptoms and physical signs which characterise the many different conditions which may produce dulness in the neighbourhood of the heart. Suffice it to say, that the observer must first endeavour to determine to which group of conditions (consolidation of the lung, fluid in the pleura, etc.), the lesion is to be referred; and having decided that point, he must next endeavour to determine the exact pathological character of the structural alteration which is present.

Step No. 3.—If the dulness is directly derived from the heart itself, does it result from increased exposure (i.e. apparent enlargement), or from actual increase in the size of the organ?

By far the most common cause of 'increased exposure' of the heart is retraction of the anterior margins of the lungs, a condition which usually results from pleurisy or cirrhosis.

In seeking then to decide whether the increased dulness is due to apparent or actual enlargement of the organ, attention must be particularly directed to the condition of the lungs. A history of previous pleurisy; the fact that the anterior margins of the lungs are fixed by adhesions, and do not expand and cover up the heart during inspiration—a point which can be determined by percussion and auscultation, during inspiration and expiration respectively; or, the presence of symptoms and signs of cirrhosis, phthisis, etc., would of course be in favour of increased exposure (apparent enlargement)—an opinion which would be confirmed by the absence of symptoms and signs of pericardial or cardiac disease, or of any extra-cardiac cause of enlargement of the heart, such as cirrhosis of the kidney or atheroma.

But while these are the points to which attention should be directed, in order to make a diagnosis, it must be confessed that a positive opinion cannot always be arrived at; and it is still more difficult to exclude any enlargement of

the heart itself, in those cases in which the increased exposure is due to forward displacement of the organ. In many of these cases the heart is actually enlarged as well as displaced; and in those cases in which there is no enlargement of the organ, the strong cardiac impulse which may be very noticeable, and the presence of intra-cardiac murmurs, which may be produced by pressure alterations at the valvular orifices, may make it impossible to exclude all cardiac hypertrophy. In cases of this description then (which, as I have previously remarked, are extremely rare), a positive opinion that the increased dulness is due to forward displacement, and not to enlargement of the heart itself, could only be ventured upon, when:—

(a) There is distinct evidence of an aneurism or tumour behind the heart, *i.e.* of the presence of an efficient cause of forward displacement.

(b) There are no signs nor symptoms of cardiac disease; and no extra-cardiac cause of enlargement such as chronic Bright's disease.

Step No. 4.—If the dulness is due to an actual increase in the size of the organ, does it result from fluid in the sac of the pericardium, or from enlargement of the heart itself?

This question, which involves the differential diagnosis of pericardial effusion and hypertrophy and dilatation of the heart, will be more appropriately considered in the detailed description of pericarditis, which will be afterwards given.

Step No. 5.—If the increased dulness is due to enlargement of the heart itself, is the enlargement general or partial, and what is the pathological character and cause of the condition?

This point, too, will be more conveniently considered under the detailed description of hypertrophy and dilatation of the heart.

DIMINISHED DULNESS OVER THE PRÆCORDIA.

Diminished dulness over the præcordia is comparatively seldom due to diminution in the size of the heart itself; it most frequently depends upon some alteration in the lungs or adjacent viscera. The chief causes of diminished dulness

over the præcordia are, in the order of their relative frequency, as follows:—

1. Increased covering up of the heart by the lungs. By far the most common cause of this condition is emphysema, but it is sometimes due to the anterior margins of the lungs being fixed over the heart by pleuritic adhesions; or to the fact, that the heart is pushed up under cover of the lungs by some gaseous, fluid or solid accumulation in the abdomen.

In some healthy persons, in whom the lungs are very voluminous, the area of absolute cardiac dulness is much smaller than usual, and may be completely abolished during a full inspiration; but these cases are easily recognised by the facts, that the area of cardiac dulness is present during a full expiration, and that there are no signs nor symptoms of cardiac or pulmonary disease.

2. Gaseous accumulations in the pleura, stomach, or intestines.

3. Gas in the pericardium.

4. Atrophy of the heart itself.

The differential diagnosis of diminished dulness over the præcordia.

There is not, as a rule, much difficulty in deciding this question. The steps which it is convenient to follow in order to arrive at a correct conclusion are as follows:—

Step No. 1.—Does the diminished præcordial dulness depend upon extra- or intra-cardiac¹ conditions?

Step No. 2.—If extra-cardiac, is the diminished dulness due (a) to some abnormal condition of the lungs; (b) to distention of the stomach and intestines; or (c) to air in the pleural cavity (pneumo-thorax)?

The observer should remember that atrophy of the heart sufficiently great to cause diminished præcordial dulness is extremely rare, and could only be diagnosed by the method of exclusion.²

¹ Under the head of intra-cardiac conditions, I include abnormal conditions of the pericardium, as well as of the heart itself.

² Atrophy of the heart is met with in many wasting affections, more particularly in cancer of the pylorus. In many cases of this kind (wasting diseases with atrophy of the heart) the lungs are emphysematous, and the diminished præcordial dulness is partly due to increased overlapping of the heart by the lungs.

In all cases, then, in which diminished præcordial dulness is met with, the practitioner should, in the first place, make a careful physical examination of the patient, paying particular attention to the condition of the lungs, stomach, and adjacent viscera. By this means he will be able to determine whether the physical signs of emphysema, pneumo-thorax, or any of the other extra-cardiac causes of diminished dulness (which I have just mentioned) are present.

When the diminished dulness is due to pneumo-pericardium, the physical signs of that condition—which are very definite, and which I shall afterwards describe—will be discovered in the course of the examination.

Should the result of this examination be negative (*i.e.* should physical examination fail to elicit any cause for the condition), he will have to ask himself whether the diminished dulness is due to a voluminous (but not emphysematous) condition of the lungs, or whether it is caused by atrophy of the heart itself. The condition of the general health; the state of the cardiac dulness during a full expiration;¹ and particularly the presence of any well recognised cause of atrophy of the heart, such as cancer of the pylorus, are the points to which attention is to be directed in order to decide this point.

AUSCULTATION.

Auscultation is an extremely important means of physical examination. It is founded upon the facts:—

(1) That during each cardiac revolution certain sounds are generated within the heart.

(2) That these sounds depend upon definite physical conditions, more especially upon the condition of the valve-flaps, and the manner in which they are closed and stretched.

(3) That modifications in these physical conditions (*i.e.* alterations in the valve-flaps, and in the manner in which they are closed and stretched) are attended by corresponding modifications in the cardiac sounds.

¹ When the lungs are voluminous, but otherwise healthy, the cardiac dulness becomes distinctly perceptible during a complete expiration. In atrophy of the heart, a full expiration does not produce such a marked effect.

By means, then, of auscultation, we are able to obtain valuable information as to the condition of the valvular apparatus of the heart; and since the mode of closure and stretching of the mitral and tricuspid valves depends, in great part, upon the condition of the walls of the left and right ventricles, and since the manner in which the aortic and pulmonary valves are closed and stretched depends upon the condition of the aortic and pulmonary blood-pressures, we have in auscultation a valuable means of ascertaining the condition of the cardiac muscle, and of the blood-pressure in the aorta and pulmonary artery respectively.

Further, by means of auscultation we can observe the rhythm of the heart, and the exact frequency of the cardiac contractions.¹ Auscultation, too, is the most important means which we possess of determining the presence of disease in the pericardial sac.

The main objects of auscultation are to ascertain:—

1. Whether both cardiac sounds are audible or not.
2. The character of the cardiac sounds, whether normal or not.
3. If abnormal, the exact nature of the modifications which are present.

But in order that these points may be thoroughly understood, and that the significance of their different modifications may be correctly appreciated, I must now describe the normal character of the cardiac sounds, their mode of production, the nature of the alterations which they undergo in disease, and the manner in which these modifications are produced.

The normal Heart Sounds and their mode of production.

During the period which elapses between the commencement of one ventricular contraction and the commencement

¹ In some cases in which the heart is acting quickly and feebly, and more especially in those cases in which some of the cardiac pulsations fail to reach the wrist, auscultation and the cardiograph are the only certain means of ascertaining the exact frequency of the cardiac contractions.

of the ventricular contraction immediately succeeding it, that is to say, during every complete cardiac revolution or cycle, two sounds are generated, which are termed respectively the first and second sounds of the heart. The two sounds are separated by periods of silence, or pauses, as they are technically termed; and the whole cardiac cycle is therefore composed of four separate parts, viz.:—

1. The first sound.
2. The first silence or pause.
3. The second sound.
4. The second silence or pause.

The *first* sound corresponds in time to the contraction of the ventricles, *i.e.* its rhythm is *systolic*; and it is mainly produced by the sudden tension of the auriculo-ventricular (mitral and tricuspid) valve-segments and their chordæ tendineæ, partly by the contraction of the muscular walls of the ventricles.¹

The *second* sound follows the contraction of the ventricles, *i.e.* its rhythm is *diastolic*; and it is produced by the closure, and, more especially, by the sudden tension of the arterial (aortic and pulmonary) valve-flaps.

Now, since the heart is a double organ, four sounds are in reality generated during each cardiac cycle, viz., two first sounds, produced by the sudden tension of the mitral and tricuspid valve-flaps (the *mitral first sound* and the *tricuspid first sounds* respectively), and two second sounds, produced by the sudden tension of the aortic and pulmonary valve-flaps (the *aortic* and *pulmonary second sounds* respectively). But in as much as the action of the two hearts is, under ordinary circumstances, perfectly synchronous, the two first sounds are synchronous, and the two second sounds are synchronous; and hence a single first sound (which is made up of the mitral and tricuspid first sounds), and a single second sound (which is made up of the aortic and pulmonary

¹ The tilting of the apex against the wall of the chest, the sudden tension of the walls of the ventricles, and the passage of the blood from the ventricles into the aorta and pulmonary artery, have also been thought to aid in the production of the first sound of the heart.

second sounds) are heard when the stethoscope is applied over the præcordia.

The relative duration of the cardiac sounds and silences.

Under ordinary circumstances, *i.e.*, when the heart is healthy and the pulse of normal frequency, each cardiac sound, and each silence, has a definite duration, which, for practical purposes, is sufficiently accurately expressed as follows:—If the whole cardiac cycle be divided into ten equal parts, the first sound will occupy four-tenths, the first silence one-tenth, the second sound two-tenths, and the second silence three-tenths of the whole. This measurement which is taken from Walshe (*Diseases of the Heart*, page 48), is a good working division, though not absolutely accurate. According to Dr Gibson¹ the average absolute duration of each phase of the entire cardiac cycle is as follows:—

Auricular Systole.	Ventricular Systole.	Ventricular Diastole.	Entire Cycle.
·112 sec.	·368 sec.	·578 sec.	1·058 sec.

This division of the cardiac cycle is graphically represented in figures 36 and 37, by reference to which the exact relationship of the cardiac sounds and silences will be more easily appreciated.

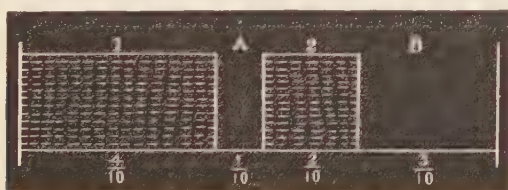


FIG. 36.—Diagrammatic representation of the cardiac cycle.

- 1=first sound.
- 2=second sound.
- A=first or short silence.
- B=second or long silence.

¹ *Journal of Anatomy and Physiology*, vol. xiv. p. 237.

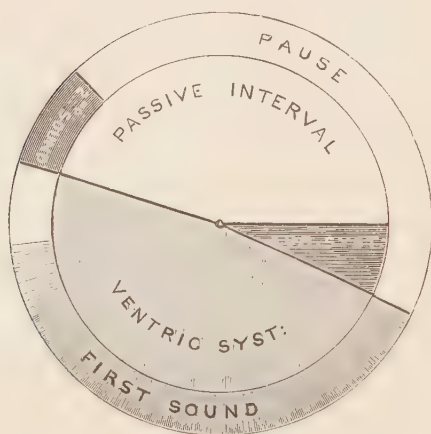


FIG. 37.—Diagrammatic representation of the cardiac cycle. (After Gairdner, modified by Sharpey).

The characteristic features of the individual cardiac sounds.

The first sound is, as we have seen, systolic, and lasts twice as long as the second, which is diastolic; but in addition to these differences in rhythm and duration, the two sounds present certain other distinctive features. The first sound is dull, muffled, and booming in character; it gives one the impression of being produced at some distance from the ear; while the second sound is sharp, abrupt, accentuated, and superficial, *i.e.* it appears to be produced close to the ear. The syllables lupp dupp give a very good idea of the normal characters of the cardiac sounds.

The characteristic features of the first sound are best heard at the apex of the heart, those of the second sound at the base.

But further, the first sound which is produced in the left heart (*i.e.* the mitral first sound) is longer and more muffled than the tricuspid first sound; while the aortic second is louder and more accentuated than the pulmonary.

The points of differential maximum intensity of the individual sounds.

In order to appreciate these differences, *i.e.* in order to hear the sounds which are generated at any one of the four

valvular orifices, apart, so far as is possible, from the other three, it is necessary to listen at certain points of the chest wall, which I am in the habit of terming the *points of differential maximum intensity of cardiac sounds and murmurs*.

The position of the points is as follows:—

Mitral sounds are best differentiated at the apex of the heart.

Tricuspid sounds at the lower end of the sternum, or rather at the junction of the lower left costal cartilages with the sternum. (Some authorities say the junction of the right lower costal cartilages with the sternum.)

Aortic sounds at the second right costal cartilage.

Pulmonary sounds at the third left costal cartilage.

The fact, that the distinctive characters of any individual sound (mitral, tricuspid, aortic, and pulmonary) are not well heard if the stethoscope is placed over the exact position of the valve at which that sound is generated, is owing to the close juxta-position of the valves. According to Walshe 'a superficial area of half an inch square will include a portion of all the four sets of valves *in situ*; an area of about one quarter of an inch, a portion of all except the tricuspid;' while Sibson states that each of the higher orifices overlaps in position the orifice immediately below it. 'Thus the pulmonic orifice at its lower and right edge is situated to a slight extent in front of the upper and left edge of the aortic orifice; the right posterior or lower flap of the aortic valve is situated in front of the upper third or two-fifths of the mitral orifice; and the lower two-thirds or three-fourths of the mitral orifice are behind the corresponding upper portion of the tricuspid orifice.'—Russell Reynold's *System of Medicine*, vol. iv. p. 86.

The exact relationship of the valves to the surface of the chest is, according to the same observers (Walshe and Sibson), as follows:—

Pulmonary.—According to Walshe¹ 'the upper or free edge of the pulmonary valves lies horizontally, and in the mass of persons, a shade above the upper edge of the third left cartilage, the body of the valve consequently a little lower than this,—the left edge of the sternum having closely the same width of the vessel on both sides.'

Aortic.—The aortic valves, according to Walshe, lie horizontally a very little further inwards, and lower than the pulmonary, corresponding to the union of the third left cartilage with the sternum.

According to Sibson the upper and left border of the aortic orifice, especially during the diastole, is situated behind the lower portion of the third cartilage near the sternum; and its lower and right border,

¹ *Diseases of the Heart*, fourth edition, p. 6.

especially during the systole, is situated behind the middle line of the sternum, on a level with the upper portion of the fourth cartilage.

Mitral.—The attached edge of the mitral valve, according to Walshe, lies almost horizontally about a quarter of an inch lower than the attached bases of the aortic valves, very slightly further inwards than these, and deeper within the chest than the tricuspid valve. The attached border lies on the level of the union of the third cartilage with the sternum, nearer, as a rule, the upper than the lower border.

According to Sibson the mitral orifice is seated behind the left half of the sternum, at the upper two-thirds of the lower third of that bone, on a level with the fourth cartilage, the fourth space, and the upper portion of the fifth cartilage.

Tricuspid.—‘The attached edge of the tricuspid,’ according to Walshe,

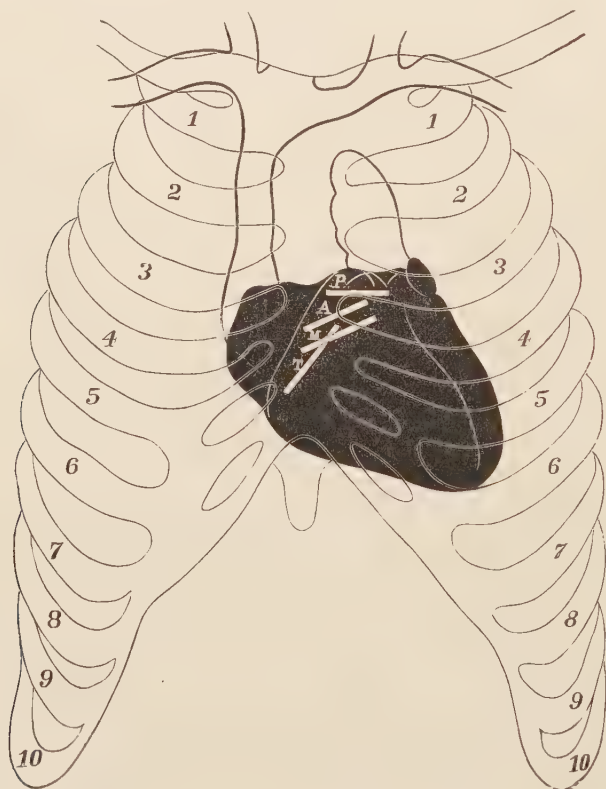


FIG. 38.—Diagram to illustrate the position of the valvular orifices. (After Gee, slightly modified.)

Note.—The aortic valve is a little too low.

‘slantingly placed across the sternum from above downwards, and from left to right, inclines from the neighbourhood of the sternal edge of the third left interspace nearly to the sternal end of the fourth right interspace, or fifth right cartilage;’ while Sibson states that in a healthy man with a well-formed chest, the tricuspid orifice is situated behind the lower fourth of the sternum to the right of the middle line of that bone, its upper border being on a level with the lower edge of the fourth cartilage, and its lower border being behind the lower end of the sternum, and the articulation to it of the right sixth cartilage.

Mode of distinguishing the two sounds of the heart.—Under ordinary circumstances there is no difficulty in distinguishing the two sounds of the heart; but in some cases of disease, more particularly when the heart is acting quickly and irregularly, and when the first sound is short and valvular, it may be very difficult to say which is the first and which is the second sound. The same difficulty is also experienced in the observation of some cardiac murmurs. Now, in both cases, attention must be particularly directed to the rhythm of the sound or murmur, and to its point of differential maximum intensity. In the case of murmurs the direction of propagation is also of importance, as I shall afterwards point out.

In order to ascertain the exact rhythm, the observer should listen over the præcordia, and, at the same time, apply the fingers of the right hand over the position of the apex beat. When the apex beat is not available, the sound should be timed by the carotid pulse. The radial pulse is not suitable for this purpose, owing to the fact that an appreciable interval elapses between the contraction of the ventricle and the occurrence of the pulse at the wrist.

Alterations in the heart sounds which occur in disease.

The alterations of the heart sounds which occur in disease are either simple modifications of the normal sounds (*quantitative* changes), or absolute alterations (*qualitative* changes).

The quantitative changes include :—

1. Alterations in the loudness or intensity, and in the duration or sound characters (tone and purity) of the cardiac sounds.

2. Alterations in the position of the points of differential maximum intensity.

3. Reduplications.

The qualitative changes embrace the various forms of endocardial and exocardial murmurs.

*Alterations in loudness or intensity.*¹

The intensity or loudness of the cardiac sounds may be increased or diminished in disease. In some cases, all four sounds (mitral, tricuspid, aortic, and pulmonary) are modified; in others, the sound produced at one valvular orifice only is affected.

But although alterations in intensity are common in disease, it must not be supposed that all variations of this description are pathological. On the contrary, we know that the loudness, of the heart sounds, varies in different individuals, even in health, and in the same (healthy) individual under different conditions; and that it is extremely difficult to fix upon an average healthy standard. When the alteration (from what we consider the average healthy standard) is considerable, when it is permanent, and more especially when some organic disease (either of the heart or other organ) capable of producing it, is present, there is no difficulty in deciding that the alteration is pathological. Another point which enables us to distinguish some of the pathological alterations is this, that in the physiological variations of intensity the relative degree of loudness of the different heart sounds is generally preserved, the aortic second sound, for instance, being louder than the pulmonary, the mitral than the tricuspid; whereas in some of the pathological variations the relative intensity of the sounds derived from the right and left hearts is perverted, the pulmonary being louder than the aortic, the tricuspid than the mitral.²

¹ Alterations in the loudness or intensity of the heart sounds are very often associated with alterations in the duration and sound characters; but for convenience of description it is better to consider each of these modifications separately.

² In many pathological alterations the relative intensity of the sounds derived from the right and left hearts is, of course, preserved.

The intensity or loudness of the heart sounds, *as heard over the præcordia*, is the resultant of two conditions, viz. :—

(1) The amount of sound, so to speak, which is generated within the heart.

(2) The facility with which the sound generated within the heart is conducted to the ear.

Variations in the intensity or loudness of the heart sounds, *as heard over the præcordia*, may therefore be due to :—

(1) Alterations in production.

(2) Alterations in conduction.

For convenience of description we may term the former *actual* and the latter *apparent* alterations.

Let us now consider, in more detail, the pathological and clinical conditions which are associated with increase and diminution of the heart sounds, *as heard over the præcordia*, respectively.

Increased intensity or loudness of the heart sounds as heard over the præcordia.

Increased intensity or loudness of the heart sounds, *as heard over the præcordia*, may, as I have previously stated, be either apparent or real.

Apparent increase.—In this condition, in which the heart sounds, are louder than in health, a normal amount of sound, so to speak, is generated within the heart, and the increased intensity, *as heard over the præcordia*, is due to the conditions for the conduction of sound, from the heart to the ear, being more favourable than in health.

Now the pathological and clinical conditions which favour increased conduction are :—

1. Abnormal thinness of the chest wall, wasting of muscle, and especially of the subcutaneous fat.

2. Increased exposure of the heart, which, as we have already seen, may be due to retraction of the anterior margins of the lungs (cirrhosis, pleurisy, etc.), or to forward displacement of the heart.

3. Consolidation of the portions of the lungs adjacent to the heart (pneumonia, phthisis, etc.).

4. The presence of a cavity, suitable for the amplification of sound, *i.e.* a thin-walled cavity in contact with the heart on the one hand and with the chest wall on the other. The cavity may be situated in the lung itself (as in phthisis); in the pleura (as in pneumo-thorax); or, it may be a dilated stomach.

5. Air in the sac of the pericardium (pneumo-pericardium). In the latter cases (4 and 5) the heart sounds usually have a metallic or amphoric character, and are sometimes auto-audible.

Real or actual increase.—In this condition the amount of sound which is generated within the heart, is above the normal healthy standard.¹

Now, since the chief causes of the normal heart sounds are the closure, and more particularly the sudden tension of the valvular segments, it follows that anything which produces more forcible closure and more sudden tension of the valvular segments will produce an actual increase. It is necessary to add, that the valvular segments must be healthy; where they are rendered rigid and incapable of vibrating, the cardiac sounds may be feeble, muffled, and impure, although the other conditions which favour the increased production of sound are present.

In some cases *all* the valvular segments are more forcibly and suddenly stretched, and all the cardiac sounds are consequently intensified. Excited action of the heart, such as results from violent muscular effort, neurotic palpitation, ex-ophthalmic goitre, etc., is the most common cause of this condition (*i.e.* of increased intensity of *all* the heart sounds).

In other cases, and these are by far the most important in a diagnostic point of view, one of the cardiac sounds only is exaggerated.

¹ When the conditions for conduction are unfavourable, the heart sounds, as heard over the *præcordia*, may be of normal, or even diminished intensity, even although the amount of sound generated within the heart is in excess.

Increased intensity of the aortic second sound occurs in all conditions in which the blood pressure, in the aorta, is above the normal (the aortic segments must, as I have previously remarked, be fairly healthy). Increased aortic tension may result from:—(a) an obstruction to the passage of blood through the arterial system, the amount of blood propelled into the arterial system (or what amounts to the same thing, the force of the cardiac pump) being normal; (b) an excessive quantity of blood being pumped into the aorta, the peripheral resistance being normal. (When the flow through the peripheral parts of the circulation is abnormally free, an excessive quantity of blood may be propelled into the arterial system without any increase of the arterial tension resulting).

The chief pathological and clinical conditions, therefore, associated with accentuation of the aortic second sound are:—

1. Obstruction to the passage of the blood through the minute arteries—a condition which occurs more particularly in chronic Bright's disease (the cirrhotic form of kidney) and in atheroma. (Dr Broadbent, Dr Mahomed, and others think that the chief cause of the obstruction in many of these cases is situated in the capillary system of vessels rather than in the minute arteries.¹)

In these cases the left ventricle is hypertrophied, and there is therefore a double cause for the increased aortic tension, viz., obstruction in front, and the powerful propelling force behind.

2. Atheroma, dilatation, and aneurism of the aorta.

3. Hypertrophy of the left ventricle.

All cases of hypertrophy of the left ventricle are not attended with an accentuated aortic second sound. In mitral regurgitation, for example, there is usually hypertrophy of the left ventricle, but in consequence of the leak through the mitral orifice, the amount of blood pumped into the aorta (by the hypertrophied left ventricle) is usually smaller than normal, the aortic tension is, therefore, diminished, and the aortic second sound decreased. Again, in aortic regurgitation, although the hypertrophied and dilated left ventricle propels an excessive amount of blood into the aorta, the aortic second sound is, in consequence of the incompetent

¹ *British Medical Journal*, Aug. 25, 1883, p. 357.

condition of the aortic valve and the altered condition of the aortic segments, usually completely absent, and replaced by a murmur.

Increased intensity of the pulmonary second sound.—Increased intensity of the pulmonary second sound occurs in all those conditions in which the blood-pressure within the pulmonary artery is in excess. And increased pulmonary tension, may, like increased aortic tension, be due either to obstruction in front, or increased pressure from behind, these two causes being very generally combined.

The chief clinical conditions associated with accentuation of the pulmonary second sound are :—

1. Mitral lesions (both stenosis and regurgitation) which produce stagnation of the blood in the lungs.

Skoda was the first to direct attention to the great importance of the accentuation of the pulmonary second sound in cases of mitral stenosis, and to point out that the 'amount of accentuation' is an index or gauge of the extent of the mitral lesion. It must, however, be remembered :—(1) that this statement only holds good so long as the right ventricle is powerful and the tricuspid valve sound. In advanced stages of mitral disease the pulmonary second sound may be less loud than at the earlier periods of the case, the explanation being either, that in consequence of the right ventricle having become feeble, or the tricuspid valve having given way, less blood is being injected into the pulmonary artery ; the blood pressure, therefore, and consequently the intensity of the pulmonary second sound, are less than at the earlier stages of the case, *i.e.* before the tricuspid began to leak ; (2) that in gauging the extent of a mitral lesion by the loudness of the pulmonary second sound, it is necessary to allow for pulmonary causes of accentuation.

2. Some lung affections, notably cirrhosis and emphysema, in which there is a primary¹ obstruction to the passage of the blood through the lungs.

3. Hypertrophy of the right ventricle. As I have previously pointed out, hypertrophy of the right ventricle is a secondary condition which results from some obstruction

Primary, as compared with the *secondary* obstruction, which is due to mitral lesions.

to the passage of the blood through the lungs. In cases of this description there is, therefore, a double cause for the increased pulmonary tension, which produces the accentuation of the pulmonary second sound, viz., obstruction in front and increased pressure behind.

Increased intensity of the mitral first sound.—When the mitral first sound is much intensified, its duration and tone are usually at the same time modified; it is shorter than normal, more abrupt, more accentuated; in fact it loses the distinguishing characters of the first sound, and assumes those of the second.

A combined condition of hypertrophy and dilatation, together with an irritable condition of the muscular fibre, attended by increased rapidity (celerity) of contraction, are the conditions which more particularly favour the production of this form of modification.

Temporary over-action of the heart (palpitation, etc.) is also a common cause of increased loudness of the mitral first sound; but in cases of this description, the other cardiac sounds are also intensified.¹

It might readily be supposed that hypertrophy of the left ventricle would produce increased loudness of the mitral first sound. Such, however, is not the case; in fact, in solid hypertrophy of the left ventricle (*i.e.* hypertrophy without dilatation), even when the segments of the mitral valve are perfectly healthy and elastic, the mitral first sound is usually weaker (more muffled) than in health (see page 154, where the cause of the muffling is explained). This fact is, I think, a strong argument in support of the valvular as against the muscular origin of the first sound.

Increased intensity of the tricuspid first sound, usually depends upon hypertrophy and dilatation of the right ventricle. The same remarks, which have just been made regarding accentuation of the mitral first sound, apply here; the right being substituted for the left ventricle, and the tricuspid for the mitral valve.

¹ The mitral segments must, for the reasons previously given (see page 144), be fairly healthy, *i.e.*, sufficiently elastic to be thrown into vibration.

The differential diagnosis of increased intensity of the heart sounds.

The recognition of the cause of the increased intensity of the heart sounds is not, as a rule, a matter of much difficulty. The steps in the inquiry are as follows:—

Step. No. 1. Are all the heart sounds intensified; or, is the accentuation limited to the sound produced at one of the valvular orifices?

Step. No. 2. If all the sounds are intensified, is the condition due to some temporary cause, such as over-action from excitement, neurotic palpitation, and the like; or, is it associated with structural changes in the heart?

The points to which attention is to be directed in order to solve this question are (a) the presence of any obvious cause of temporary over-action or excitement; (b) the condition of the heart after a sufficient time has elapsed to allow of any temporary over-action from nervousness, excitement, etc., to have subsided; and especially (c) the presence or absence of any indications of organic change (such as increased dulness on percussion, etc.) in the heart itself.

Step No. 3. If the accentuation is confined to the sound produced at one of the valvular orifices, what is its cause?

When the aortic second sound is accentuated, attention must be particularly directed to the condition of the urine; the state of the superficial vessels; the presence or absence of pressure symptoms within the thorax, and the physical examination of the aortic arch; for renal disease, atheroma, and aneurismal dilatations of the aorta, are, as we have previously seen, the most common causes of this condition, *i.e.* accentuation of the aortic sound. It is important too, to note the condition of the left ventricle; for hypertrophy of the left ventricle is another cause of increased loudness of the aortic second sound.¹

¹ The hypertrophy of the left ventricle, which is associated with accentuation of the aortic second sound, is usually secondary to some form of arterial obstruction. Hypertrophy due to mitral and aortic valve lesions is not (for the reasons previously given) usually attended by increased intensity of the aortic second sound,

When the pulmonary second sound is accentuated, attention must be particularly directed to the condition of the mitral valve, for mitral lesions are the most common (cardiac) cause of the condition. Should the mitral valve be healthy, the lungs¹ must be carefully examined, and the presence or absence of hypertrophy of the right ventricle must be noted.

When the mitral or tricuspid first sounds are intensified, the condition of the left and right ventricles must be particularly investigated, and the causes of hypertrophy and dilatation looked for.

Diminished intensity or loudness of the heart sounds as heard over the præcordia.

Diminished intensity or loudness of the heart sounds, as heard over the præcordia, may be either *apparent* or *real*.

Apparent diminution.—In this condition, in which the heart sounds, *as heard over the præcordia*, are less loud than in health, a natural amount of sound, so to speak, is generated within the heart, and the diminution is due, either to (a) the conditions for conduction being less favourable than in the normal state, or to (b) the heart sounds being obscured by other sounds produced within the chest, *e.g.* bronchitic râles.²

Now the chief pathological and clinical conditions which interfere with conduction are:—

1. Excessive thickness of the chest-wall more particularly of the subcutaneous fat.
2. Abnormal over-lapping of the heart by the lungs—a condition which reaches its highest degree of development in pulmonary emphysema.
3. Fluid in the sac of the pericardium.

Actual diminution.—In this condition the amount of

¹ We have previously seen that accentuation of the pulmonary second sound is almost always due to obstruction to the flow of blood through the lungs, and that this condition may be *primary*, *i.e.* due to primary alterations in the lungs, cirrhosis, emphysema, etc., or *secondary*, *i.e.* the result of mitral lesions.

² In these cases it may be impossible to hear the heart sounds although the conditions for conduction are favourable.

sound which is generated within the heart, is less than normal. The diminution may be due to the following conditions :—

1. Less forcible closure of the valve-segments.
2. Less sudden closure of the valve-segments.
3. Structural alterations in the valve-segments, which produce loss of elasticity, or interfere with vibration.¹

In some cases, *all* the heart sounds are weakened. In others, the sound produced at *one* of the valvular orifices only, is affected.

The clinical and pathological conditions associated with diminished intensity of all the cardiac sounds are :—

1. Feeble action of the heart resulting from temporary conditions, such as syncope, and the structural changes which affect the cardiac muscle in the course of an attack of continued fever, notably in typhus.

2. Feeble action of the heart resulting from permanent structural changes, such as fatty and fibroid degeneration, great dilatation of the ventricular cavities, etc.

The conditions which produce diminished intensity of the individual heart sounds are as follows :—

Diminished intensity of the aortic second sound.—Diminished blood-pressure in the aorta is the usual cause of this condition ; but it may result from rigidity of the aortic cusps.²

The following *pathological and clinical* conditions are the usual causes of diminished blood-pressure within the aorta,

¹ In many of these cases the sound is impaired or replaced by a murmur.

² Diminished intensity of the aortic second sound, due to this cause, is seldom observed. It probably could only occur in those cases in which the rigidity was considerable ; and, when the rigidity of the cusps is great, the aortic valve is very often incompetent, and the aortic second sound replaced by a murmur. It must, too, be remembered that forcible tension (distention) of the base of the aorta probably also takes part in the production of the aortic second sound.

It is important too, to remember, that rigidity of the aortic segments is often associated with general atheroma and dilatation of the aortic arch ; and that in those conditions the aortic second sound instead of being enfeebled is usually accentuated. If the aortic segments and base of the aorta were so rigid as to be incapable of acting as good sound producers, accentuation would not, of course, be observed, even where there was atheroma and dilatation of the aortic arch ; but such a degree of rigidity is uncommon.

and hence of diminished intensity of the aortic second sound:—

1. All structural changes which impair the 'driving' power of the left ventricle, such as fatty and fibroid degeneration of the cardiac muscle, dilatation, etc. In these cases the amount of blood propelled into the arterial system is insufficient to keep it properly distended, and the aortic blood-pressure is consequently below the normal.

2. Mitral lesions (both stenosis and incompetence.) Here again the amount of blood discharged by the left ventricle into the aorta is below the normal quantity.¹

Diminished intensity of the pulmonary second sound may result from muscular weakness or dilatation of the right ventricle, or from disease of the tricuspid orifice. These conditions are almost always secondary either to mitral lesions or to primary lung affections. Diminished intensity of the pulmonary second sound is seldom, therefore, primary.

It usually happens that the pulmonary second sound is accentuated in the earlier periods of these cases; and it is only after the secondary dilatation of the right ventricle has become considerable, or after the tricuspid valve has given way, that the accentuation disappears, and diminished intensity of the pulmonary second sound is observed.²

Diminished intensity of the mitral first sound.—Weakness of the wall of the left ventricle, whether temporary or permanent, is a fertile source of diminished intensity of the mitral first sound. In many of these cases the wall of the right ventricle is affected in the same manner, and the sounds of the right heart are also weakened.

The condition may also be due to the fact, that the valve-segments are less suddenly stretched than in health. This is probably one cause of the 'muffling' of the first sound which

¹ We have previously seen that the pulmonary second sound is accentuated in mitral stenosis. The contrast, therefore, between the aortic and pulmonary second sounds, is great, and is due to a double cause, viz., diminution of the aortic and increase of the pulmonary.

² In most of these cases the diminished intensity is only relative as compared with the marked accentuation in the earlier periods. It is seldom that the pulmonary second sound is less distinct than in the average run of healthy persons.

is seen in solid hypertrophy of the left ventricle. In consequence of the obstruction in front (either at the aortic orifice itself or in the arterial system beyond) the contraction of the left ventricle is more deliberate than in health, and the tension of the mitral segments is more gradually effected.

Some authorities have supposed that in cases of this description, the left ventricle, in consequence of being over distended, has a greater difficulty in obtaining a 'grip' of the blood, so to speak, than it has in health;¹ and that this is the explanation of its slow and gradual contraction. I am, however, disposed to doubt the correctness of this explanation. 'Loss of grip' would be much more likely to occur in dilatation than in solid hypertrophy; and dilatation of the left ventricle, as we have already seen, tends rather to produce accentuation, at the same time that it shortens the duration of the first sound.

Others have explained the diminished intensity of the mitral first sound which is associated with some cases of hypertrophy, by supposing that the 'initial tension' of the mitral valve is so great that the contraction of the ventricles does not produce the usual amount of tension, and therefore of vibration of the valve-segments. In other words, in consequence of the excessive intra-ventricular pressure during diastole,—a condition which we may suppose is produced by the regurgitant current in the case of aortic incompetence—the segments of the mitral valve are closed, and partly stretched, at the end of diastole, *i.e.* before the ventricular systole occurs.²

¹ Solid hypertrophy always results from some obstruction in front. The hypertrophy which occurs in mitral and aortic regurgitation is always associated with some dilatation.

² Increased 'initial tension' could only occur in aortic regurgitation, or in mitral lesions in which the blood pressure in the left auricle during diastole was increased. Now, in mitral regurgitation the first sound is replaced by a murmur; and in mitral stenosis, the diminution of the first sound—which is certainly present—is, I believe, due partly to the fact that the mitral segments have lost their normal elasticity, partly to the small amount of blood which the left ventricle contains, and therefore to the diminished force with which the mitral segments are closed and stretched.

Another reason, which has been advanced as a partial cause for the diminished intensity of the first sound in cases of solid hypertrophy is, that the sound which is produced by the tension of the mitral and tricuspid valves is less easily conducted through the thick muscle of the ventricular wall.¹ The correctness of this explanation is, in my opinion, doubtful.

Structural alterations which impair the elasticity of the valve segments are also the cause of diminished intensity of the mitral first sound. This is probably one of the conditions which produce the modified first sound which occurs in mitral stenosis, to which I have already alluded.

Diminished intensity of the tricuspid first sound.—Impairment of the force of the right ventricle (fatty degeneration, fibroid degeneration), and dilatation of the ventricular cavity, are the chief causes of this condition.²

Alterations in the duration of the heart sounds.

Alterations in duration are only noticeable in connection with the first sound. It may be either longer or shorter than in health.

Increased duration of the first sound is generally associated with the slow and deliberate contraction which is seen in solid hypertrophy, more especially with the solid hypertrophy which is caused by stenosis of the aortic orifice. In addition to the alteration in duration the sound is, in these cases (as we have previously seen) more muffled than in health.

Diminished duration of the first sound is generally due to dilatation, or dilatation and hypertrophy of the left ventricle, in fact, it occurs in all conditions in which the ventricular wall is enfeebled. There is often too, in such cases, an irritable condition of the cardiac muscle, which produces increased celerity of contraction. These alterations are frequently met with in chlorosis and fatty heart.³

¹ *Diagnosis of Diseases of the Heart*, by Dr Sansom, page 104.

² In these cases tricuspid regurgitation often occurs, and instead of a weak first sound in the tricuspid area, we have a tricuspid systolic murmur.

³ In the more advanced stages of chlorosis the mitral first sound may be replaced by a systolic murmur.

Alterations in the tone of the heart sounds.

Alterations in tone are often combined, as we have already seen, with alterations in intensity. The chief modifications in tone are as follows:—

1. *The heart sounds may be more muffled than in health.*¹

Muffling of the first sound may be due to:—(a) impaired conduction, the clinical causes of which have been already detailed (see page 149); (b) weakness of the walls of the left ventricle from fatty or fibroid degeneration, etc.; (c) solid hypertrophy of the left ventricle; and (d) alterations in the segments of the mitral valve, which impair its elasticity, but which do not give rise to regurgitation.

Muffling of the aortic second sound generally results from loss of elasticity in the aortic segments, and is often associated with atheroma of the base of the aorta and disease of the coronary arteries.

Impure heart sounds.—The term impurity is given to a modification of the heart sounds, which is closely allied to muffling on the one hand and murmur on the other. In fact an impure sound may be described as a sound which has lost its normal well-defined character, which is usually of diminished intensity, and which is somewhat muffled and murmur-like. An impure sound may often be transformed into a murmur by exciting the heart to more vigorous action—making the patient walk quickly up and down the room, ascend a stair, etc.²

An impure sound, then, is suggestive of valvular imperfection; and the presence of an impure aortic second sound, more especially,³ may be a physical fact of great practical

¹ Alterations of this description are chiefly important in connection with the mitral and aortic sounds.

² I must caution the observer against over-exciting the heart in those cases in which he desires to produce or intensify a cardiac murmur, and particularly against applying any sudden strain, or producing any sudden rise of arterial blood-pressure. An American traction machine, which I lately saw in the consulting room of a well-known London physician, seems to me especially dangerous, and very likely to produce rupture of a thin-walled aneurism, were such a lesion present.

³ An impure first sound is of much less importance, for slight incompetence at the mitral or tricuspid orifices is frequently due to temporary and curable conditions.

importance. (Impurity of the aortic second sound generally depends, as we have previously seen, upon rigidity of the aortic cusps; and rigidity of the aortic cusps is often associated with atheroma of the base of the aorta and disease of the coronary arteries,—conditions which are frequently suspected, but which it may be impossible to detect by means of physical examination. Now, in more than one case of angina-like pain in the chest which has come under my own personal observation, the presence of an impure aortic second sound was the only physical alteration which could be detected, and as we shall afterwards see, when I come to treat of angina pectoris, the recognition of disease at the root of the aorta is, in suspected cases of that disease, a point of great practical importance.)

2. *The heart sounds may be more accentuated than in health.*

The conditions, which produce modifications of this description, have already been considered, and need not again be detailed (see p. 143).

3. *High-pitched, metallic, auto-audible heart sounds.*

When the conditions for conduction are extremely favourable; when, for instance, that portion of lung, which covers the heart and great vessels, is consolidated or retracted, the heart sounds may be unusually loud and high-pitched. A loud and high-pitched pulmonary second sound, for example, is met with in some cases of pneumonia and pleuro-pneumonia, and probably depends, as Quincke was the first to point out, upon retraction or consolidation of the portion of lung which overlaps the root of the pulmonary artery.

When a cavity, well suited for the reproduction and conduction of sound, is in contact with the heart or great vessels on the one hand, and with the chest wall on the other, the heart sounds may have a metallic or amphoric character. In some of these cases,¹ and in some cases of consolidation of the

¹ A striking example of auto-audible heart sounds, depending upon the presence of a cavity in the lung, is described by Dr Smith in the *Lancet* for December 18, 1880.

lung, the heart sounds may be auto-audible, *i.e.* heard by the patient himself, and by the physician without applying the ear to the chest.

A striking example of auto-audible heart sounds depending upon consolidation of the lung came under my observation during the summer of 1881. A patient who for some weeks had been suffering from a limited pleuro-pneumonia of the left base, was suddenly seized with acute pain in the left side, which was followed by quick pulse, elevation of temperature and rapid consolidation. The day following the occurrence of the attack, dulness on percussion and tubular breathing were present over the lower two-thirds of the left lung, and *the heart sounds could be readily heard, when the patient sat up, without applying the ear to the chest.*

Reduplication of the Heart Sounds.

We have previously seen that although four sounds (two first and two second sounds) are generated within the heart, during each cardiac revolution, two only (a single first and a single second sound), are, under ordinary circumstances, heard over the præcordia, and that the blending of the mitral and tricuspid first sounds and of the aortic and pulmonary second sounds is due to the fact, that the action of the two ventricles is usually synchronous.

Now, under certain conditions, even in health, and not unfrequently in disease, two first sounds or two second sounds are audible; and the heart sounds are said to be *reduplicated*, or, as Dr Barr prefers to term it, *duplicated*.

The exact cause of this doubling or reduplication has given rise to great debate, but most authorities are agreed—and with their opinion I entirely concur—that the condition is, in the great majority of cases, due to a synchronic closure of the mitral and tricuspid valve flaps; in short, that when the first sound is reduplicated, its two component parts, *viz.*, the mitral and tricuspid first sounds, are individually audible; and that when the second sound is reduplicated, both the aortic and pulmonary second sounds are separately heard.

It will be advisable, however, to consider the individual reduplications, their clinical significance, and probable causation a little more in detail.¹

Reduplication of the First Sound.

Reduplication of the first sound is rare—much rarer than reduplication of the second sound—a circumstance which is probably due, as Dr Barr has suggested, to the fact that the first sound is produced by several different factors, and that it is a sound of considerable duration. In order that the first sound may be perceptibly reduplicated, *i.e.* that the mitral and tricuspid first sounds may be separated by a perceptible interval, it is necessary to have a considerable degree of asynchronism between the closure and tension of the aortic and pulmonary valve flaps—a more considerable asynchronism than is required to produce perceptible doubling of the second sound.

Further, I am of opinion that reduplication of the first sound can only occur when—in addition to asynchronism—the duration of one or other, or of both of its component parts (*i.e.* of the mitral and tricuspid sounds) is lessened, or when the duration of the whole cardiac cycle is very considerably increased (*i.e.* when the pulse is slower than in health).² Under ordinary circumstances the first sound

¹ While I am of opinion that asynchronism is the cause of the reduplication in most cases, I am not prepared to assert dogmatically that it is so in all. Some cases are with difficulty explained by this view, and for them, some of the many other theories which have been advocated, possibly hold true. Space does not permit me to consider the arguments for and against these various theories in detail. Those readers who are interested in the subject should consult the writings, more especially of Drs Barr and Sansom, where they will find full details.—(See articles in the *Medical Times and Gazette* of January and February 1877, and in the *Liverpool Medico-Chirurgical Journal*, July 1882, by Dr Barr; and *Diagnosis of Diseases of the Heart*, by Dr Sansom, p. 106, *et seq.*)

² Asynchronism, without any diminution in the duration of the mitral and tricuspid first sounds, might account for the condition, if the duration of the whole cardiac revolution (*i.e.* of the diastolic portion of the revolution) was much increased; if, for example, the heart were contracting 30 instead of 72 times in a minute,

occupies (roughly) $\frac{4}{10}$ ths of the entire cardiac revolution. (See fig. 39.) Now, granting that the tricuspid first sound—as is

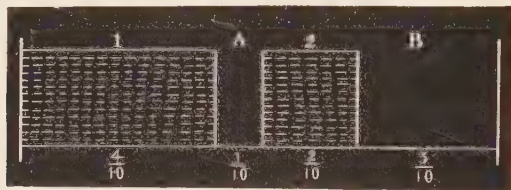


FIG. 39.—Diagrammatic representation of the cardiac cycle.

1=first sound.

2=second sound.

A=first or short silence.

B=second or long silence.

probably the case—is of somewhat shorter duration than the mitral first sound, say that it occupies $\frac{3}{10}$ ths of the entire cardiac revolution, it is almost impossible to conceive that reduplication could be produced by asynchronism alone, that is to say, that reduplication could occur in any case in which the mitral and tricuspid first sounds and the whole cardiac cycle (the cycle for both the right and the left hearts) preserved their normal length. For if such were the case, $\frac{4}{10}$ ths + $\frac{3}{10}$ ths, *i.e.* $\frac{7}{10}$ ths of the entire cardiac revolution, would be occupied by the reduplicated first sound, and to this we must add the interval which is required, *i.e.* which must separate the two component elements of the reduplicated sound, in order that each element may be perceived as a separate and distinct sound by the ear. Further, I may add, that reduplication of the first sound can never occur when the heart is acting quickly.

The three conditions, then, which I believe to be necessary, in most cases, for perceptible reduplication of the first sound are—

(1) Considerable asynchronism in the contraction of the two ventricles.

(2) Diminished duration of one or other or both of the component parts of the reduplicated sound.

(3) Slow action of the heart.

Numerous other theories have been suggested in order to explain reduplication of the first sound, viz. :—

1. That it is due to non-synchronous tension of the individual segments of the auriculo-ventricular valves, owing to the absence of perfect uniformity of the contraction of the papillary muscles.—(*Guttmann.*)

2. That the reduplication is due to splitting up of the first sound into its component parts of ventricular impulse and valve tension.—(*Hayden.*)

3. That it is due to a double mitral valve click, the true mechanism of which remains to be discovered.—(*Professor D'Espine.*)

4. 'That the contraction of a dilated, and especially of a hypertrophied left auricle, becomes sonorous, and that the first division of the double first sound is the result of the auricular systole.'—(*Dr George Johnson.*)

5. 'That it is due to a presystolic being closely followed by a normal first sound, the presystolic sound being produced by the sudden floating upwards of the mitral curtains occasioned by the auricular systole.'—(*Dr Sansom.*)

Dr Barr has, I think, conclusively disposed of these various theories, with perhaps the exception of the last, in the article to which I have already referred.—(*Liverpool Medico-Chirurgical Journal*, July 1882.)

Dr Sansom's theory is the only other one, in addition to that adopted in the text, which seems to me at all probable. He explains its mechanism as follows :—'During the interval immediately succeeding the relaxation of the ventricle, the blood, subject to the tension in the left auricle and pulmonary veins, has been pouring into the ventricular cavity; the fluid naturally finds its way in the direction of least resistance, that is, its course, when impelled towards the apex, is round the walls of the ventricle, thus coming *behind* the curtains of the mitral valve, and bellying them out (so to speak) as the sails of a ship are bulged by the force of the wind. At the moment of auricular systole, the ventricle, as yet only partially full, is rapidly distended, the force of contraction of the auricle giving an impulse to the apex of the ventricle, and, as may of course be inferred, giving a *contre-coup* to the already partially strained mitral curtains. In normal conditions such *contre-coup* is inaudible, but when the auricle is more than ordinarily powerful, or when the mitral valve is so changed as to give rise to the sound of membranous tension, it becomes perceptible closely preceding the sound produced by the ventricular systole—that is, the sound of complete closure of the valves guarding both auriculo-ventricular orifices plus the muscular sounds of the ventricles.'—(*Diagnosis of Diseases of the Heart*, p. 115.)

Doubling of the first sound is said to occur physiologically at the end of expiration or the commencement of inspiration; and pathologically (though rare) it has been met with in various conditions, amongst which the following are

the chief:—functional disorders of the heart; diseased (degenerated) conditions of the cardiac muscle; hypertrophy of the ventricles; dilatation of the ventricles; valvular lesions, more especially mitral and tricuspid lesions.

As I have already stated, asynchronic closure of the mitral and tricuspid valves is, in my opinion, the most important cause of the condition; and we must now consider how the asynchronism may be brought about.

The first sound of the heart is probably composed, as we have previously seen, of the sounds which are generated by:—(a) the sudden closure and tension of the mitral and tricuspid valve flaps; (b) the contraction of the ventricles; and (c) the impulse of the apex beat against the wall of the chest;¹ and all of these conditions immediately result from the contraction of the muscular walls of the ventricles, which is, in its turn, due to the rhythmical discharge of motor nerve force in the cardiac ganglia. Further, we have seen that the ganglia are stimulated, to discharge, by the presence of blood under a certain tension in the cardiac cavities, and that the action of the ganglia can be held back (inhibited) or accelerated by impulses passing to the heart through the pneumogastric and sympathetic nerve trunks.

Now, the theory which explains the reduplications of the heart sounds by asynchronic closure of the valve flaps necessarily presupposes that the action of the right and left ventricles is to some extent at least independent, and that the one ventricle may begin or may end contraction before the other.²

¹ The sudden tension of the valve flaps and chordæ tendineæ is probably by far the most important factor of the three.

² In reference to this part of the subject Dr Barr says: 'In reply to the objection which has been urged against the possibility of asynchronism, on account of the interlacement of the cardiac fibres and the observed consentaneousness of the ventricular action, I say that I have seen, felt, and heard asynchronous action, and so have no difficulty in admitting the possibility of its occurrence. I do not say that such asynchronism as exists between the auricles and ventricles is possible between the two ventricles, but I do say that each side of the heart has its own proper muscular fibres, as well as those which are common to both sides; and those proper fibres form in great part the deepest layers, and so are first

Theoretically, therefore, we may suppose that reduplication of the first sound may be due either to:—(A) such accelerated or retarded action of the left ventricle as is sufficient to cause the first sound, which is generated within the left heart, to be separated by an interval, sufficient to be appreciated by the ear, from the first sound, which is generated within the right heart; or to (B) such accelerated or retarded action of the right ventricle that the same result—an appreciable interval between the first sounds of the right and left hearts—may occur.¹

Further, we may presume that accelerated or retarded action of either ventricle may be due to:—

1. Differences in the pressure of the blood in the two ventricles by reason of which the muscular fibres of one ventricle are stimulated to contract before the muscular fibres of the other.

It is, I think, highly improbable that this condition alone is ever the cause of a reduplicated first sound—for, as we shall see when I come to speak of reduplication of the second sound, increased pressure of the blood in either ventricle, generally depends upon obstruction in front; and in such conditions, although the increased blood pressure may stimulate the affected ventricle (*i.e.* the ventricle which has to cope with the difficulty, and in which the increased pressure occurs) to commence contraction, yet by reason of the obstruction which it has to overcome, it is longer in emptying itself, and in such cases reduplication of the first sound does not of course occur.

subjected to the stimulating influence of distention in producing contraction; and in those fibres which are common to both sides, it is possible for the wave of contraction to commence, as it were, at one extremity of the fibre and be propagated to the other. Each side has more or less independently its own nerve supply, its own peristaltic action, and notwithstanding that both sides are set to the same time, and that there is a complex interlacement of fibres, yet it is quite possible, nay, it is an experimental fact, that one side may begin or end contraction before the other.’—(*Liverpool Medico-Chirurgical Journal*, July 1882, p. 206.)

¹ I have already expressed the view that modifications in the duration of the component elements of the reduplicated sound, or of the duration of the whole cardiac cycle, are also necessary.

2. Alterations of the nerve apparatus of one ventricle, by reason of which the motor ganglia of one side are rendered more or less irritable than those of the other.¹

Such a condition may possibly explain the reduplications of the first sound, which are said occasionally to occur in some functional (neurotic) affections of the heart.

3. Structural alterations of the muscular fibres of one ventricle, by reason of which its contraction is more or less quickly effected than that of the other ventricle. In some cases of fatty degeneration (in the early stage at least) a condition of 'irritable weakness' is sometimes met with, in which the muscular fibre is more easily excited, but in which its contraction is less sustained (*i.e.* of shorter duration) than in health. It is easy to conceive, therefore, that if such a change were confined to the muscular fibre proper to one ventricle, or were much more marked in one ventricle than in the other, that the first sound produced in the affected side might anticipate that produced in the normal one.

So again in cases of fibroid degeneration, the contractility of the affected ventricle (granting that the change were confined to one ventricle) might, we can easily suppose, be unduly delayed, and therefore sufficient asynchronism to cause reduplication of the first sound, produced.

Reduplication of the Second Sound.

Reduplication of the second is much more frequent than reduplication of the first sound. It occurs as a physiological condition at the end of inspiration and commencement of expiration; and as a pathological phenomenon it occurs in many different conditions, amongst which stenosis of the mitral valve, and primary obstruction to the flow of blood through the lungs (such as is produced by cirrhosis, emphysema, etc.) are the chief.²

¹ If Dr Gaskell's view—that the rhythmical action of the heart is due to a rhythmical property possessed by the cardiac muscle itself, independently of any ganglionic nerve apparatus—is correct, the explanation given under this head (2) will not of course hold good.

² Reduplication of the second sound occurs in at least one-third of all the cases of mitral stenosis, and is highly suggestive of that condition.

As I have previously stated, I agree with those authorities who believe that the cause of reduplication of the second sound is asynchronous closure (and tension) of the aortic and pulmonary valve flaps; and we may theoretically suppose that the asynchronous closure may result from the fact that the systole of either ventricle is so accelerated or so delayed as to allow of an appreciable interval between the recoil of the aorta and pulmonary artery, *i.e.* between the production of the aortic and pulmonary second sounds.

Further, we may suppose that the following conditions may produce such asynchronous contraction of the ventricles as will cause reduplication of the second sound:—

1. Derangement of the nerve apparatus of the heart, by reason of which the motor ganglia¹ of one ventricle are rendered more irritable or less irritable than those of the other. This is probably the cause of the reduplicated second sound which is not uncommon in functional (neurotic) affections of the heart.

2. Structural alterations of the muscular fibres proper to either ventricle, by reason of which they are rendered either more irritable or less irritable than in health; and by reason of which the contraction of one ventricle is, therefore, unduly accelerated or unduly retarded. (See remarks on page 161.)

3. Differences in the pressure of the blood in the two ventricles, by reason of which the action of one is accelerated.

Some authorities, who allow that asynchronous closure, of the aortic and pulmonary valves, is the cause of the reduplication of the second sound in mitral stenosis, have supposed that, in consequence of the increased blood-pressure, which is present in the right chambers of the heart, the right ventricle is first stimulated, that it first completes its systole, and that the first element in the reduplicated sound is consequently pulmonic. But with this opinion I do not agree, for, as Dr Barr points out, it does not necessarily follow that the

¹ If Dr Gaskell's view—that the rhythmical action of the heart is due to a rhythmical property possessed by the cardiac muscle itself, independently of any ganglionic nerve apparatus—is correct, the explanation given under this head will not of course hold good.

ventricle, which is first thrown into action, will first complete its systole; for the duration of ventricular systole is not a fixed quantity. Further, we know that (within the range of its working power) the duration of the ventricular systole varies directly with the amount of resistance, which has to be overcome; in other words, the greater the resistance, the longer the systole. (Witness the slow deliberate pulse of aortic stenosis.) And many good clinical observers positively state that the accentuated element of the reduplication is the second element, and that the accentuated or second element is heard over the area of the pulmonary artery and not of the aorta, *i.e.* that it is pulmonary.

4. Differences in the resistance in front, by reason of which the systole of one ventricle is rendered of shorter or of longer duration than that of the opposite one.

I agree with Dr Barr and others, who think that this is the true explanation of the reduplicated second sound in mitral stenosis—that although the right ventricle is, by reason of the increased blood-pressure in the right cavities of the heart, first stimulated to contract, yet, in consequence of the obstruction to the passage of the blood through the lungs, and the difficulty which the ventricle has in emptying itself, its systole is so prolonged, that a sufficient amount of asynchronism is produced to allow of an appreciable interval between the aortic and pulmonary sounds; in other words, that the reduplication is primarily due to alterations in the blood-pressure of the aorta and pulmonary artery respectively, and the consequent alteration in the duration of the systole of the right and left hearts; the systole of the left ventricle being, in consequence of diminished aortic pressure, shorter, while the systole of the right ventricle is, by reason of increased pulmonary pressure, longer than in health.

Numerous other theories have from time to time been advocated to explain the reduplication of the second sound, amongst which the following may be mentioned :—

1. That the second element of the reduplication is produced by a sudden tension of the mitral curtains after the normal second sound

has occurred. Dr Sansom, whose theory this is, gives the following explanation of it :—‘ It seems to me, therefore, that reduplication of the second sound, in cases of mitral stenosis, can be best explained thus :— The first element of the reduplication is the normal second sound; the tension in the aorta being feeble, it is the *pulmonic* element which has the chief share in the production of such second sound. The second element of the reduplication is the sudden tension of the abnormal mitral-curtains produced after the relaxation of the left ventricle. I am not prepared to say that systole of the auricle is *essential* to produce this sudden tension; it may be quite possible that the reaction of the distended pulmonary veins and left auricle may be sufficient to cause it. So it may occur previously to the auricular contraction, the latter occasioning or reinforcing the presystolic murmur, separated by a slight interval from the second element of the reduplication.’

Dr Sansom illustrates his theory, the only one in addition to that adopted in the text which seems to me probable, by the following diagram :—

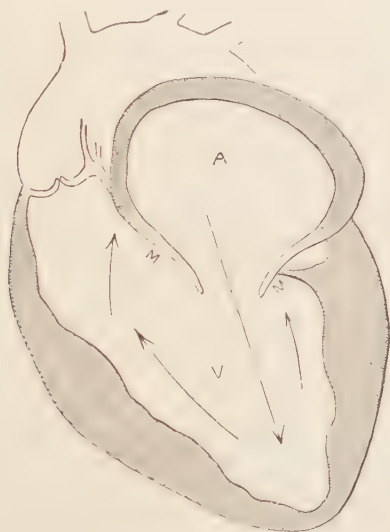


FIG. 40.—Diagram illustrative of the mechanism of pseudo-reduplication of the heart sounds. (After Sansom).

Ideal section through the left auricle (A) and left ventricle (V); M.M., mitral curtains; the arrows show the direction of the blood current during the ventricular diastole and auricular systole.

¹ *Diagnosis of Diseases of the Heart*, p. 120.

2. Asynchronous closure of the individual segments of one valve, aortic or pulmonary.—(Guttmann.)

3. That the first element of the reduplication is the ‘audible snap which attends the systolic closure of the valve segments, separated, by an appreciable interval, from the diastolic snap (of the arterial systole).’—(Dr George Balfour, *Lancet*, March 5, 1881, p. 396.)

4. That the reduplication of the second sound originates at the narrowed orifice itself; and that the two elements of the phenomenon in question, which form a sound which is always more or less muffled and impure, are in reality the component parts of a murmur.—(Guttmann—*Hand-Book of Physical Diagnosis*, p. 279.)

5. That the second element of the reduplicated sound is the sound which is produced by the contraction of the dilated and hypertrophied left auricle.—(Guttmann, *Handbook of Physical Diagnosis*, p. 279.)

ALTERATIONS IN THE POSITION OF THE HEART SOUNDS.

The heart sounds are occasionally heard over parts of the chest at which, under ordinary circumstances, they are inaudible or only faintly heard. As a rule, such sounds are less loud than the sounds heard over the heart itself; but this is not always the case, and it occasionally happens that the abnormally placed sound, so to speak, is actually louder than the heart sounds heard over the præcordia.

The conditions which give rise to these alterations in the position of the heart sounds are as follows:—

1. Anything which renders the tissues and parts surrounding the heart better conductors of sound, so that the heart sounds are conveyed to parts of the chest at which, under normal circumstances, they are inaudible or only faintly heard. Consolidations of the lung, tumours in the mediastinum, and curvatures of the spine, are some of the clinical conditions which are included under this head.

2. Changes in the size and position of the heart itself. When, for instance, the left ventricle is enlarged, the apex is displaced downwards and to the left, and the heart sounds are, so to speak, displaced with it. So, again, when the heart is pushed to the right by an extensive left-sided pleuritic effusion, the heart sounds may be inaudible under the left, but loudly heard under the right nipple.

3. Aneurisms of the thoracic aorta. When the heart sounds are loudly heard over a part of the chest at which, under normal circumstances, they are faint or inaudible, and when the abnormally placed sound is situated over the course of the aorta, the presence of an aneurism should be suspected and the other symptoms and signs of that condition carefully looked for. In some cases of this description the heart sounds, more especially the second, are very loud (*i.e.* accented), over the seat of the tumour.

ABSOLUTE MODIFICATIONS OF THE HEART SOUNDS.
MURMURS.

The alterations of the heart sounds which I have hitherto described were *simple* or *quantitative* in character; the modifications which we must now consider are *absolute* or *qualitative*. In other words, the normal heart sounds are either partly or entirely replaced by new sounds, which, in normal conditions (*i.e.* in conditions of perfect health and during tranquil action of the heart) are never heard over the præcordia. To these qualitative modifications the term *murmur* is given.

Cardiac murmurs, *i.e.* new sounds heard either in the place of or along with the heart sounds, may be produced either outside the heart or in the heart itself. To the former the term *exo-cardial*, to the latter *endo-cardial*, is applied.

Exocardial Murmurs.

Strictly speaking the term *exo-cardial murmur* should include all murmurs produced outside the heart, but as a matter of fact it is usually limited to those murmurs, in reality, friction sounds, which are synchronous with the action of the heart, and which are produced either in the sac of the pericardium or in the adjacent pleura, *i.e.* to those *exo-cardial* murmurs which are heard over the præcordia. Arterial and venous murmurs, which are, of course, also *exo-cardial*, are classed as distinct. It is in the usually accepted—the narrow and restricted sense—that I shall make use of the term.

Exocardial murmurs, then, are friction sounds, which are synchronous with the contractions of the heart; usually double (to-and-fro sounds); generally having the harsh, grating character which friction sounds commonly possess; as a rule heard loudest over the centre of the heart, *i.e.* over the anterior surface of the right ventricle, or at the base of the organ; almost always intensified, and sometimes altered in character by the pressure of the stethoscope; and which are produced, in the great majority of cases, by the rubbing together of the inflamed and roughened surfaces of the pericardium. Exocardial murmurs are, therefore, almost invariably, indicative of pericarditis.¹

Quite exceptionally a friction sound, synchronous with the action of the heart, and resembling very closely (it may be exactly) the to-and-fro friction murmur of pericarditis, is produced outside the sac of the pericardium by the rubbing of the roughened outer surface of the pericardium against the front wall of the chest or adjacent surface of the pleura; and to this sound the term *pericardial-pleural-friction murmur* is applied.²

Endocardial Murmurs.

Endocardial murmurs, *i.e.* murmurs which are produced within the heart itself, are either *organic* or *functional*. Under the former head are included all those cases in which the murmur depends upon distinct structural alterations (usually of the valvular mechanism) which can be recognised after death. Under the latter term, *i.e.* functional murmurs, it is customary to describe those cases in which the murmur depends upon temporary and curable conditions, or in which no distinct alteration of the valvular mechanism can be

¹ Exocardial friction murmurs do not bear such an exact relationship to the sounds of the heart as endocardial murmurs. They are, however, as stated in the text, synchronous with the cardiac contractions—a point which at once distinguishes them from ordinary pleural friction murmurs.

² The manner in which the pericardial-pleural-friction murmur is to be distinguished from the ordinary friction murmur of pericarditis will afterwards be described. (See p. 327.)

detected after death. The distinction is one of great practical utility, but it is not strictly accurate from a pathological point of view. We have previously seen, *firstly*, that mitral and tricuspid regurgitation—conditions which are attended by well marked murmurs—frequently result from ‘relative’ or ‘muscular’ incompetence, the valve segments being perfectly healthy; and *secondly*, that the defective muscular action which produces the incompetence, depends in some cases upon temporary and curable, in others upon permanent and incurable, conditions. Now, in both cases, the murmur is undoubtedly due to structural alterations, which, provided that the patient were to die while the murmur were still audible, could be recognised on the *post-mortem* table. In both cases, therefore, the murmur is, strictly speaking, organic. Provided that this fact is kept in view, it is a matter of practical convenience and utility to describe the curable cases as functional, and the incurable as organic.

Before we consider each of these conditions (organic and functional murmurs) in detail, it may perhaps be well to describe the manner in which murmurs are produced.

Mode of production of Endocardial Murmurs.

Endocardial murmurs are due to the production, within the heart, of vibrations which can be perceived by the ear of the observer; and most authorities are agreed that the audible vibrations, which are represented externally as murmurs, are generally (some say are always) due to the production of sonorous fluid-veins in the blood itself. According to M. Chauveau, these sonorous fluid-veins are always generated when the blood stream passes, *with sufficient force* from a part of the circulatory system which is actually or relatively constricted, into a part which (in comparison with the part behind it) is actually or relatively dilated; as, for instance, when the blood current is forcibly driven through a constricted mitral orifice into the ventricular cavity beyond.

Other authorities suppose that the blood current in its passage through the heart may throw the tissue itself, over which it passes, into vibration; and this would appear to be the

manner in which Dr Austin Flint supposes that the presystolic murmur of mitral stenosis is generated.

The vibration of a filament or tongue of lymph in the blood current is also supposed to be the cause of some auto-audible and musical murmurs.

Audible vibrations or murmurs may probably also be produced by the collision of two opposing blood currents; when, for instance, the blood stream which is flowing backwards through an incompetent mitral orifice meets the blood stream which is advancing into the left auricle from the pulmonary veins.

The fact that a certain intensity of blood current is necessary for the production of a murmur is a point of great practical importance. We frequently find, for example, that a murmur, which depends upon organic and permanent conditions (*e.g.* the presystolic murmur of mitral stenosis) disappears as the case progresses, the dilated and weakened auricle (in the case of mitral stenosis) being no longer able to produce a blood wave capable of generating sound. In cases of this description, and indeed in others, in which the conditions for the production of a murmur are not as yet very perfectly developed, we can, by increasing the force of the heart's action, either temporarily by exertion (walking about, ascending a stair, etc.), or by the use of cardiac tonics (*digitalis* for example), frequently convert an impure or ill-defined sound into a murmur, and can thus obtain important diagnostic evidence as to the nature of the case.

It is important, too, to remember that the composition and quality of the blood seem to exercise some influence in the production of murmurs. In conditions of anæmia, for example, venous murmurs are almost invariably present, and are without doubt due to the abnormal condition of the blood.¹

¹ Dr George Balfour, in speaking of the mode of production of the venous hum says, 'As a venous murmur is often to be heard in certain positions, such as the torcular Herophili, where no other change is at all probable, under all the circumstances of the case, except one involving an altered relation of the blood to its containing vein, we can have no hesitation in ascribing its production to that cause. And further, from what we know of the mode in which fluid veins are formed, we can have no difficulty in saying that this altered relation of the blood

It is possible, too, that the systolic murmurs which are so commonly heard over the base of the heart,¹ in conditions of anæmia, are in part at least due to the same cause. But to this point I will presently recur.

Organic Endocardial Murmurs.

Organic endo-cardial murmurs usually depend upon some defect in the valvular mechanism of the heart; occasionally they are due to congenital imperfections and malformations; very exceptionally to the presence of clots or vibrating filaments of lymph in the cardiac cavities. Organic endo-cardial murmurs are in the adult usually heard over the mitral or aortic orifices, since organic lesions of the valves, which are their most frequent cause, are usually left-sided; but they may be produced at any of the cardiac orifices. They are either *direct* or *indirect*, that is to say, they may be produced either by the blood current as it passes onwards in its natural forward (direct) course, or by a blood-current passing back-

to the walls of the vein must consist in the production of increased friction between the two, so that in those positions where there is normally a relative constriction, insufficient, however, to produce sonorous veins, the increase of friction between the wall of the vein and the layer of blood next it practically narrows the opening sufficiently to do so, by retarding the exterior portion of the blood current and leaving the central or axial portion uninfluenced. Physical laws leave no doubt as to this. It is for physiologists to explain whether it depends simply upon a watery condition of the blood or upon some other cause, and their explanation would no doubt be of the greatest importance for practical medicine.'—*Diseases of the Heart*, Second Edition, p. 170.

¹ As we shall presently see, Dr Balfour is of opinion that the basic murmurs heard in the earlier stages of anæmia are due to mitral regurgitation. But he also admits, that in the later stages, arterial murmurs may be developed, and he hints at least that the altered condition of the blood may play some part in their production. He says, for instance—'Shortly after the appearance of the primary hæmic murmur, a tricuspid murmur and jugular undulation are found to be developed. This is naturally accompanied by a pulmonary and also by an aortic systolic murmur, the active cause in the production of both these murmurs being the large blood waves sent on by the dilated and hypertrophied ventricles.'—*Ed. Medical Jour.*, Oct. 1882, p. 295. 'There is also an abnormal friction between the spanæmic blood and the walls of the blood-vessels, hence a rise in arterial blood-tension, hence also the formation of fluid veins at certain favourable points within the venous lumen, and at these points we hear the primary chlorotic murmur—the venous hum.'—*Loc. cit.*, p. 293.

wards through an incompetent valvular orifice, *i.e.* by a regurgitant or indirect current.

The points to be observed in the Clinical Investigation of Cardiac Murmurs are as follows :—

1. The rhythm of the murmur.
2. Its point of differential maximum intensity.
3. The direction in which it is propagated.
4. Its sound characters (loudness, tone, pitch, duration, etc.)

THE RHYTHM OF THE MURMUR.

By the rhythm of the murmur we mean its exact relationship to the sounds and silences of the heart. The murmur should, if possible, be 'timed' by the apex-beat, and failing this, by the carotid pulse. When the heart is acting quickly, it may be very difficult or even impossible to 'time' the murmur correctly. The difficulty of determining the exact rhythm is also increased where more than one murmur is present, and this is more particularly the case where a presystolic is combined with a systolic mitral murmur; the difficulty being less in the case of double aortic murmurs, for in such cases the interval between the systolic and diastolic portions of the murmur is usually quite distinct.

Endocardial murmurs may occur either during the systole or the diastole of the heart, *i.e.* of the ventricles; and it will now be necessary to consider each of these different murmurs, and the conditions which may produce them, in detail.

Systolic Murmurs.

Systolic murmurs correspond to the contraction or systole of the ventricles; they more or less completely replace the first, or systolic sound of the heart, and may extend into the short silence. (See fig. 41.)

They may be produced either in the left or right heart, and may be either functional or organic. They represent one or other of the following conditions:—

1. *Regurgitation through the auriculo-ventricular orifices.*—(Mitral and tricuspid regurgitation.) Both of these condi-

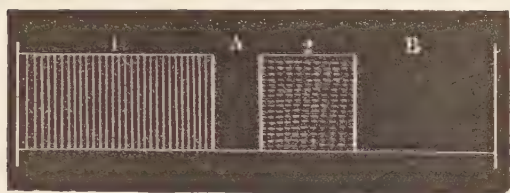


FIG. 41.—Diagrammatic representation of a systolic murmur, replacing the whole of the first sound. The murmur in this and succeeding figures is represented by continuous vertical lines, the normal sound by interrupted horizontal lines. 1=systolic murmur.

tions are common. The former (mitral regurgitation) is often primary; the latter (tricuspid regurgitation) is usually secondary, and due to disease of the mitral valve, or to (primary) obstruction to the passage of the blood through the lungs, such as occurs in cirrhosis, emphysema, etc.

Now we have seen that auriculo-ventricular regurgitation may result from :—

(a) Organic changes in the valve segments, such as puckerings, contractions, adhesions, ruptures, ulcerations; these organic changes being rare on the right side, *i.e.* at the tricuspid orifice.

(b) Muscular incompetence, *i.e.* imperfect closure of the valve in consequence of defective muscular contraction, the valve segments being healthy.

(c) Relative incompetence, *i.e.* dilatation of the valvular orifice, the valve segments being healthy.

And since both 'muscular' and 'relative' incompetence (which are often present in the same case) may depend on temporary and curable conditions, it follows that auriculo-ventricular regurgitation may be either organic or functional.

2. *The formation of a fluid vein*, or the production of sonorous vibrations in the tissues *at the arterial orifices* (aortic or pulmonary orifices), or in the first part of the arterial systems (*i.e.* in the commencement of the aorta or pulmonary artery).

The pathological conditions associated with these basic systolic murmurs are :—

(a) Constriction of the arterial orifices (aortic and pulmonary stenosis). Aortic constriction is common, pulmonary constriction extremely rare. The former is usually acquired, the latter almost always congenital.¹

(b) A tongue of lymph adhering to the arterial (aortic or pulmonary) valve flaps. This is not a common cause of basic systolic murmurs, but it does sometimes occur. The murmur which it produces may be musical and auto-audible. It may disappear in the course of treatment.

(c) Anæmia. Basic systolic murmurs are common in anæmia. They are sometimes heard in the pulmonary, sometimes in the aortic area. Their exact significance and mode of production, more especially of the systolic murmurs heard in the pulmonary area, have given rise to much debate; but to this question—which is still far from settled—I shall presently recur. (See page 188.)

(d) Dilatation of the ascending portion of the aortic arch. In cases of this description the aortic orifice is usually diseased (constricted). A murmur may, however, be produced as the blood passes from a naturally sized aortic orifice into a dilated aorta provided that it is propelled with sufficient force to generate an audible fluid vein.

It is obvious, therefore, that systolic murmurs produced at the aortic and pulmonary orifices may, like those produced at the mitral and tricuspid orifices, be either functional or organic.

Diastolic Murmurs.

Diastolic murmurs occur during the ventricular diastole. There are at least three distinct varieties, viz. :—

1. The diastolic murmurs, which occur during the period of the second sound, and which more or less completely replace it. These murmurs may, and often do, extend into the second or long pause. (See figs. 42 and 43.)

¹ In some cases of ulcerative endocarditis the pulmonary valve is affected, and a systolic pulmonary murmur, representing pulmonary stenosis, is observed. Such cases are, however, extremely rare.

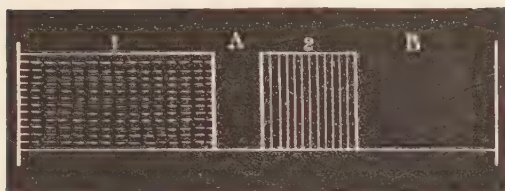


FIG. 42.—Diagrammatic representation of a diastolic murmur, replacing second sound. 2=murmur.

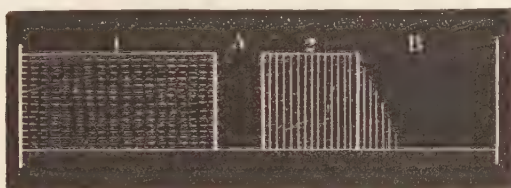


FIG. 43.—Diagrammatic representation of a diastolic murmur, replacing second sound, and extending into long pause. 2=murmur; B=long pause.

Now, since the second sound of the heart is due to the closure and tension of the aortic and pulmonary valve flaps it follows, that a murmur which replaces the second sound must represent regurgitation through the aortic or pulmonary valvular orifices. And since, as a matter of practical experience, we know that pulmonary regurgitation hardly ever occurs, a diastolic murmur, which replaces the second sound at the base, is, for practical purposes, pathognomonic¹ of aortic regurgitation. Further, we know, as a matter of practical experience, that this murmur is almost invariably organic. Aortic regurgitation generally results from organic changes in the aortic segments (contractions, puckerings, adhesions, ruptures, ulcerations), though it is occasionally due to dilatation of the base of the aorta, the valve segments being

¹ In one or two cases a diastolic murmur has been produced in an aneurism independently of any regurgitation through the aortic valves. Such a condition is, however, so extremely rare, that for practical purposes the statement in the text—that a diastolic basic murmur indicates aortic regurgitation—may be safely relied upon.

perfectly healthy, *i.e.* it is occasionally due to a condition of 'relative incompetence' of the aortic orifice.¹

2. The murmurs which occur during the latter part of the long silence or pause, *i.e.* immediately before the systole of the ventricles, and to which the term *presystolic* is usually given. (Fig. 44.)

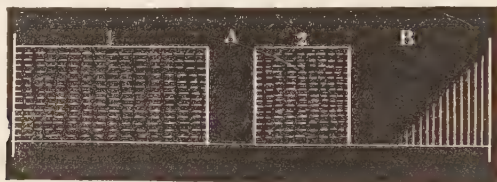


FIG. 44.—Diagrammatic representation of a presystolic murmur.

The presystolic murmur stops abruptly with the systole of the ventricle, *i.e.* with the occurrence of the first sound of the heart—a fact which is at once explained when it is understood that the murmur is produced by the formation of a fluid vein as the blood is forcibly driven by the contraction of the auricle through a stenosed mitral orifice.²

Almost all authorities are agreed in thinking that the presystolic or auriculo-systolic murmur (as Professor Gairdner, who was one of the first to direct attention to its true significance, prefers to term it), is always due to organic changes, *i.e.* to stenosis of the mitral or tricuspid orifices. And since tricuspid stenosis is very rare, a presystolic or auriculo-systolic murmur, in the great majority of cases, indicates mitral stenosis.

The stenosed condition is usually due to adhesion of the cusps, the chordæ tendinæ are often thickened, and some-

¹ This condition is (?) always associated with organic changes at the root of the aorta; but it is quite possible, as Dr George Balfour points out, that a dilated aorta may contract, and so the condition of incompetence be removed. 'Relative incompetence' of the aorta depends then upon organic changes, but it may sometimes disappear under treatment.

² In some cases of presystolic murmur, *i.e.* of mitral constriction, the first sound of the heart is replaced by a systolic murmur, and the presystolic therefore passes into the systolic murmur (*i.e.* is continuous with it), the two murmurs being as a rule distinguishable by their sound characters.

times, as it were, fused into a mass with the cusps and papillary muscles. The stenosis is sometimes due to calcareous deposits in the base of the valve and in the valve segments; occasionally a mass of vegetations obstructs the orifice; in rare cases the stenosis is caused by the pressure of a tumour, the valve segments being perfectly healthy.

Professor Austin Flint differs from the generally accepted opinion, viz., that a presystolic murmur is invariably organic. While granting of course that the murmur is generally due to mitral stenosis, he thinks it by no means pathognomonic of that condition. He believes that it may occur independently of any mitral lesion, and he has recorded several cases in support of his view. In all these cases there were aortic lesions permitting of regurgitation; and the question at once suggests itself, whether the presystolic murmur heard at the apex was produced at the mitral orifice at all. Might it not have been the aortic murmur heard at the apex of the heart? The explanation which Professor Flint gives of the production of the murmur in such cases is as follows:—‘With regard to this specimen, consider the physical conditions in life, at the instant when the auricular contraction took place. The left ventricle was filled with blood from the current passing from the auricle to the ventricle, through an unobstructed orifice, by gravitation, and in addition by the regurgitant current from the aorta. As a consequence the mitral curtains were floated away from the ventricular walls, and were not only approximated but in absolute contact. Recollect the physiological experiment by which it is shown that the mitral valve may be completely closed by injecting liquid into the left ventricle through the mitral orifice. These conditions existing, the auricle contracts and forces an additional quantity of blood into the ventricle. This mitral direct current passes between the valvular curtains which are in apposition, and throws them into vibration precisely as the lips are made to vibrate with the breath.’¹

3. The murmurs which occur during the first part of the

¹ *Lancet*, Jan. 27, 1883.

long pause, but which follow, usually with a distinct and appreciable interval, the second sound. (See fig. 45.)

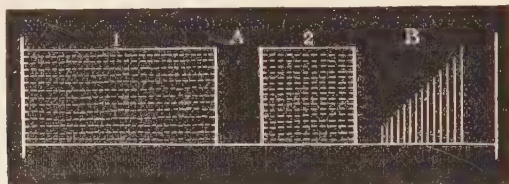


FIG. 45.—Diagrammatic representation of post-diastolic murmur, which occurs in some rare cases of mitral stenosis. It is separated from the commencement of the first sound by an appreciable interval.

This, which is by far the rarest form of diastolic murmur, is occasionally met with in mitral stenosis, and is doubtless produced by the blood which has been pent up in the left auricle and parts behind it, during the ventricular systole, *i.e.* while the mitral valve was closed, being propelled with sufficient force through the stenosed orifice to produce a fluid vein, *i.e.* to generate a murmur. Possibly too the negative pressure in the ventricle, *i.e.* the suction which occurs when the ventricle relaxes, aids in the production of the murmur.¹

The period at which the murmur occurs, *i.e.* during the first part of the long pause, clearly shows that the contraction of the auricle, which does not occur until the end of the long pause, takes no part in its production.

Combination of Murmurs.—In many cases more than one murmur is present. (See figs. 46, 47, 48.) An aortic systolic murmur is very often combined with an aortic diastolic murmur, and a mitral presystolic murmur with a mitral systolic. Further, in advanced cases of aortic regurgitation, the mitral valve often becomes incompetent, and a systolic mitral murmur is heard in addition to the aortic diastolic murmur which was present from the first. Again, in the

¹ Goltz and Gaule believe that the negative pressure appears at the beginning of the diastole, and that it is caused by the expansion of the ventricle. 'Were this the case,' says Prof. Michael Foster, 'the ventricle might be regarded not only as a force pump driving blood into the arteries, but also as a suction pump drawing blood from the auricles and great veins.'—*A Text Book of Physiology*, Fourth Edition, p. 152.

advanced stages of mitral lesions, tricuspid regurgitation frequently results from the secondary changes which are established in the right heart; while in cases of anæmia, systolic murmurs are sometimes heard at all the cardiac orifices

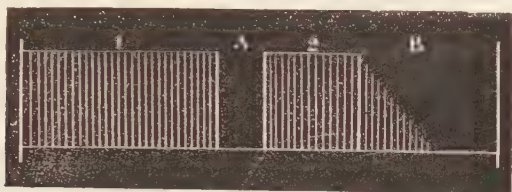


FIG. 46.—Diagrammatic representation of systolic and diastolic murmur.

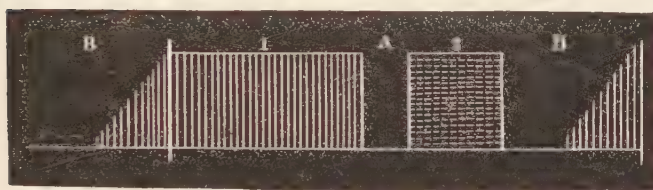


FIG. 47.—Diagrammatic representation of systolic and presystolic murmurs.

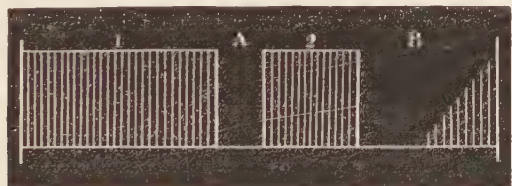


FIG. 48.—Diagrammatic representation of systolic, diastolic, and presystolic murmurs.

It is sometimes doubtful whether a cardiac sound is replaced or only obscured by a murmur, and in the case of all murmurs it is desirable, if possible, to determine whether any cardiac sound still remains. Now these points can, as Gendrin was the first to point out, sometimes be ascertained by removing the ear a short distance from the end of the stethoscope, or by so placing the instrument that the ear-piece only partly covers the external auditory meatus. By this means the *sound* is rendered *more audible*, while the *murmur* becomes *less distinct*.

The fact, that the heart sound is heard, as well as the

murmur, shows, more especially in the case of diastolic aortic murmurs, that the valvular segments, or some of them, can still be closed and put upon the stretch, in fact, that they are still capable of generating the second sound. It does not however follow that in such cases the lesion is a trivial one. In fact, in some cases in which one valve segment only is affected (as in a case which I shall afterwards relate), the contrary holds good. The fact, then, that some sound remains, suggests that some of the valve segments are still sufficiently healthy to generate the normal sound. When the disease has developed slowly, as in atheromatous disease of the valve, it is probably a favourable indication, and shows that the valve is not, as yet, very seriously affected. In acute cases—such as traumatic rupture of a valve segment, or ulceration of the segments—it may be of no value whatever, but this point will be more fully discussed in treating of the prognosis of aortic regurgitation.¹

THE POINT OF DIFFERENTIAL MAXIMUM INTENSITY OF THE MURMUR.

From what I have previously stated in speaking of the differential maximum intensity of the normal heart sound (see page 138), it will readily be understood that in order to ascertain the valvular orifice at which the murmur is produced, it is necessary to ascertain its point of differential maximum intensity. These points are shown in the diagrams (figs. 49 to 54), and are as follows:—

Mitral Murmurs.—The point of differential maximum intensity of mitral murmurs is the left apex-beat, wherever it may happen to be, not necessarily in the fifth left inter-space, an inch and a half below, and slightly internal to the left nipple, *i.e.* the position of the apex-beat in health. (See fig. 49.) Presystolic mitral murmurs are often best heard

¹ The observer must of course satisfy himself that the sound which remains is generated at the affected valvular orifice. In the case of an aortic diastolic murmur, for instance, a second sound, due to closure of the pulmonary flaps, might be heard in some situations along with the diastolic murmur, and might be referred to the aortic orifice.

slightly internal to or just above the position of the apex-beat, rather than at the apex-beat itself. (See fig. 50.)

Aortic Murmurs.—The point of differential maximum intensity of aortic murmurs is the second right costal cartilage at its junction with the sternum (the aortic cartilage). A diastolic aortic murmur may however be heard loudest at the lower end of the sternum, in consequence of the fact that it is propagated downwards in the course of the blood current which produces it. (See figs. 51 and 52.)

Pulmonary Murmurs.—The point of differential maximum intensity of pulmonary murmurs is the third left costal cartilage at its junction with the sternum (the pulmonary cartilage). (See fig. 53.)

Tricuspid Murmurs.—The point of differential maximum intensity of tricuspid murmurs is the lower end of the sternum, or rather the junction of the lower left cartilages with the sternum. (See fig. 54.) Some authorities place the point of differential maximum intensity for tricuspid murmurs at the junction of the lower *right* costal cartilages with the sternum.

A systolic murmur is sometimes heard equally loud at the base and at the apex of the heart, and it may be difficult or impossible to determine, from its mere loudness, at the two points, whether it is generated at the aortic or at the mitral orifice, or at both, *i.e.* whether there are two separate murmurs. The question must be decided in the following manner:

Firstly. The murmur must be carefully traced from one point to the other—from the apex to the aortic cartilage, for example, in the case we are supposing. If the intensity of the murmur diminishes at a point midway between the two positions (*i.e.* the two points of differential maximum intensity), there are probably two murmurs.

Secondly. The tone of the murmur at the two points must be carefully noted. If the tone differs there are probably two murmurs.

Thirdly. The direction of propagation must be observed.

If the murmur is well propagated, both upwards over the aorta, and outwards and upwards towards the axilla, there are in all probability two murmurs.

THE DIRECTION IN WHICH THE MURMUR IS PROPAGATED.

Murmurs are propagated chiefly in two ways, viz. :—(1) By *conduction*, *i.e.* through the structures in which they are generated, and by the parts which surround those structures. (2) By *convection*, *i.e.* carried by fluid in the direction in which it is flowing. As a matter of practical experience and observation, we know that the different murmurs which represent different valvular lesions, are propagated in certain definite directions, which are as follows :—

Mitral regurgitant (systolic) murmurs :—These murmurs are propagated upwards and outwards towards the left axilla (see fig. 49); and, when organic, are often well heard at the inferior angle of the left scapula. According to Naunyn,

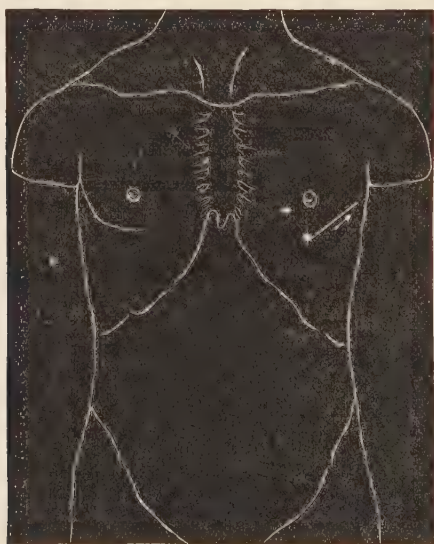


FIG. 49.—Outline figure showing point of differential maximum intensity (*) of the systolic mitral murmur (mitral regurgitation); and the direction in which it is propagated. The cross +; which is supposed to represent the normal position of the apex-beat, is placed a little too high.

Balfour, and others,¹ the systolic murmur, due to mitral regurgitation, is sometimes heard in the region of the pulmonary artery, being conducted to that part of the chest through a dilated left auricular appendix.

Mitral direct (presystolic) murmur.—This murmur is usually heard over a very limited area. The blood current, which produces it, is flowing directly towards the apex (or very nearly so) of the heart. (See fig. 50.) We can readily understand therefore why the presystolic murmur is usually limited to the position of the apex beat.

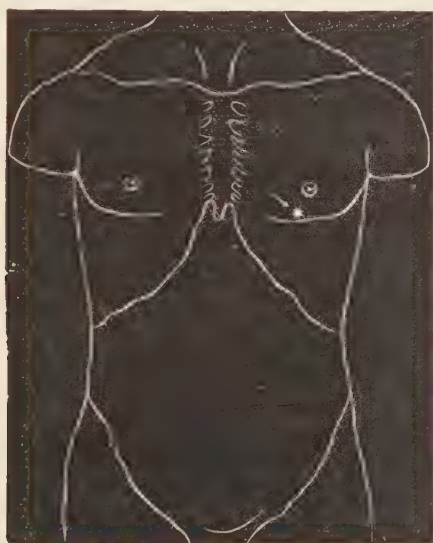


FIG. 50.—Outline figure showing point of differential maximum intensity (*) of the presystolic mitral murmur (mitral stenosis); and the direction in which it is propagated. (The murmur is often best heard a little above and internal to the apex-beat, which in the diagram corresponds to the star.*)

Aortic regurgitant (diastolic) murmurs are carried downwards by the blood current which produces them, towards the apex of the heart; but in consequence of the fact that the sternum is such a good conductor of sound, these

¹ These writers say that the murmur is not heard exactly over the pulmonary area, but a little outside it, viz., at a spot an inch and a half outside the sternum, in the third interspace, just at that spot at which the tip of the left auricular appendix comes forward from beneath the cover of the pulmonary artery.

murmurs are well conducted down that bone, and are often heard very distinctly at the xiphoid cartilage, as represented in fig. 51.

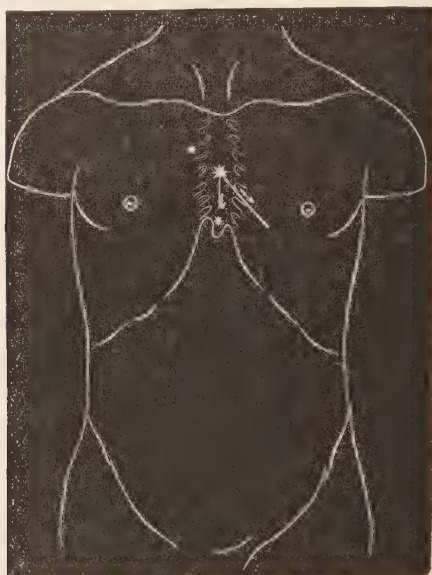


FIG. 51.—Outline figure showing point of differential maximum intensity (*) of the diastolic murmur (aortic regurgitation); and the direction in which it is propagated.

Aortic direct (systolic) murmurs are carried upwards over the course of the aorta, and the great branches which arise from it. Organic murmurs are more extensively carried in these directions than functional, *i.e.* anæmic murmurs. (See fig. 52.)

Pulmonary regurgitant (diastolic) murmurs are so extremely rare, that for practical purposes they may be almost ignored, they are propagated downwards towards the lower end of the sternum.

Pulmonary direct (systolic) murmurs, which, when organic, are extremely rare, are propagated upwards and outwards, over the course of the pulmonary artery. As a rule they cannot be traced for any distance over the surface of the chest. (See fig. 53.)

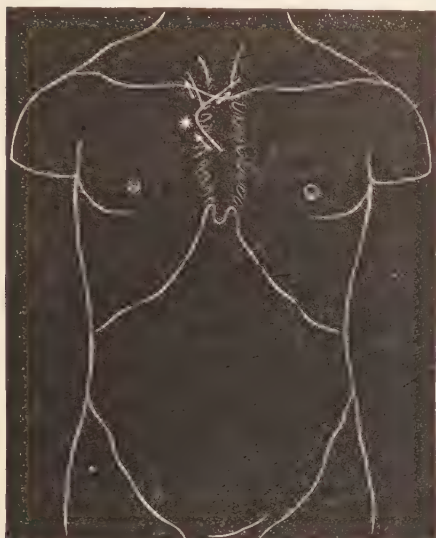


FIG. 52.—Outline figure showing point of differential maximum intensity (*) of the systolic aortic murmur (aortic stenosis); and the directions in which it is propagated.

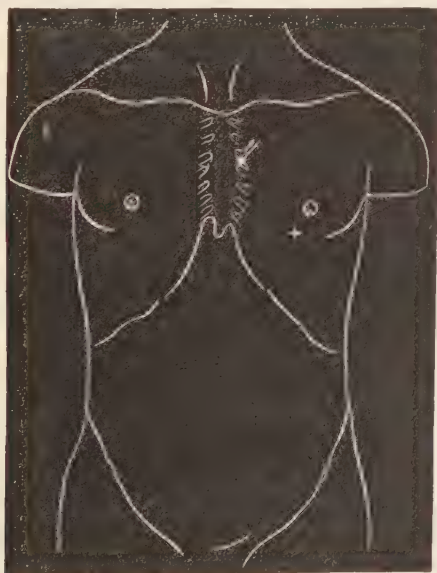


FIG. 53.—Outline figure showing the point of differential maximum intensity of the systolic pulmonary murmur, and the direction in which it is propagated.

Tricuspid regurgitant (systolic) murmurs are not, in my experience, extensively propagated over the chest, but are usually heard over a limited area. The direction of their propagation is probably that shown in fig. 54.

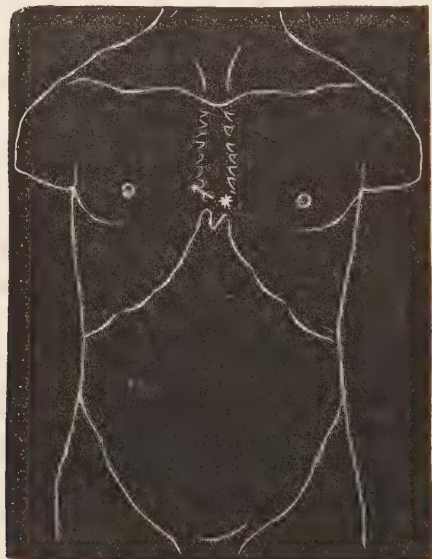


FIG. 54.—Outline figure showing point of differential maximum intensity (*) of the systolic tricuspid murmur.

Tricuspid direct (presystolic) murmurs are extremely rare, they would probably be propagated towards the apex of the heart.

THE SOUND CHARACTERS OF THE MURMUR.

Some murmurs are soft and blowing; others are harsh (grating, rasping, filing, sawing, etc.) in character; others again are whistling, cooing, and musical. Occasionally, as I have before mentioned, a murmur is auto-audible.

Inorganic murmurs are usually soft; 'direct' murmurs (*i.e.* murmurs due to obstruction at one or other of the cardiac orifices) are usually harsher and rougher than 'indirect' ones (*i.e.* murmurs due to regurgitation). But speaking generally, it may be said that the mere sound character of

the murmur is not a point of much practical importance,¹ either from a diagnostic or a prognostic point of view; and this is readily understood when it is remembered that the sound characters of the murmur, more especially its loudness, are largely due to the force of the blood current which produces it; in other words, the loudness of the murmur depends to a great extent upon the condition of the cardiac muscle. When the muscular wall of the heart is weakened or degenerated, the murmurs which are generated are faint and soft; and in advanced cases, as, for instance, in extreme cases of mitral stenosis, no murmur may be generated at all. And since a weakened and degenerated condition of the heart muscle may be due, either to temporary and curable, or to permanent and incurable conditions, it follows that the softness and faintness of the murmurs, which are produced by its contraction, cannot be relied upon as any indication of the severity of the lesion.

When, however, a harsh murmur gradually becomes fainter and softer, we may, as a general rule, conclude that the strength of the muscular wall is diminishing, and we may be pretty sure that this is the case, when the symptoms are at the same time increasing in severity. A faint murmur, then, associated with severe symptoms, is usually indicative of a grave lesion.

FUNCTIONAL MURMURS.

It is customary to include under the term functional murmurs, all those cases in which the murmur depends upon temporary and curable conditions, or in which no distinct alteration in the valvular mechanism (it would perhaps be more correct to say in the valvular segments) can be detected after death. Functional murmurs are always systolic in time, and are generally heard at the base of the heart, most commonly to the left of the sternum, in the second interspace, *i.e.* in the region of the pulmonary artery. They are sometimes also heard in the aortic, mitral, and tricuspid areas.

¹ The presystolic murmur is a notable exception. In typical cases the sound characters of the murmur (as I shall afterwards describe in detail) are so distinct as to distinguish it from all other cardiac murmurs.

The exact significance and mode of production of the functional murmurs, which are audible at the base of the heart, have given rise to great debate, and are among the most unsettled points in cardiac pathology.

Walshe divides the so-called functional murmurs into two great groups, which he respectively terms hæmic and dynamic. Under the former he places 'murmurs originating within the heart and dependent upon an unnatural state of the blood;' while under the latter he includes 'murmurs which result from abnormal action on the part of the heart,' the proper valvular mechanism being healthy. And this division indicates the two great causes of functional murmurs, viz.:— (1) *defective muscular action* allowing of mitral and tricuspid regurgitation; and (2) an *altered condition of the blood*, which probably aids, at least, in the production of the pulmonary and aortic murmurs, which are met with in conditions of anæmia.

But it will be necessary to consider the subject in more detail. Anæmia, more particularly those forms of anæmia (such as chlorosis, progressive pernicious anæmia, etc.) in which there is defective formation of blood, is the chief clinical condition in which functional endocardial murmurs occur. Now, in cases of this description the muscular fibre of the heart becomes fatty, the cardiac cavities become dilated, and the weight of the heart also becomes increased; in fact, there is a condition of combined fatty degeneration, dilatation, and hypertrophy, dilatation being, however, much in excess of hypertrophy.¹

Such a condition of the heart is just such as we would expect to produce 'relative incompetence;' and, as a matter of fact, all careful clinical observers are, I presume, agreed that, in cases of this description, mitral and tricuspid regurgitation do ultimately occur. All observers are also, I suppose, at one in thinking that arterial murmurs (pulmonary and aortic murmurs) are usually present in *advanced* condi-

¹ These changes are seen in the human subject in cases of progressive pernicious anæmia; and they have been shown by Beau to occur in the lower animals after venesection.

tions of anæmia. The point which is still undecided, and which has lately given rise to so much controversy, is the nature and significance of the basic murmur, which is heard in the second left interspace, in the *earlier* stages of anæmia. Three theories, all of which have warm supporters, have been advanced to account for the condition, viz.—

1. *That the murmur is pulmonary.*—The exact manner in which the murmur is supposed to be produced in the pulmonary artery, has not been very clearly defined by the supporters of this view; the *sudden* propulsion of a *large* blood wave of *abnormal* (spanæmic) composition into the vessel, which is probably in some cases at least dilated, seems to me an efficient cause for its production. We know, as a matter of fact, that the heart, in cases of chlorosis, is not only weak, but that it is unusually irritable; that even in the earlier stages the right ventricle is to some extent dilated; and that the blood is markedly altered in composition.

2. *That the murmur is due to mitral regurgitation*, and that it is conducted to the anterior wall of the chest through the *dilated appendix* of the left auricle.—(Dr George Balfour's theory).

3. *That the murmur is produced in the pulmonary artery* as the result of *constriction* of that vessel; the constriction being caused by the pressure of the *dilated left auricle*.—(Dr Russell's theory.) Dr Russell further believes that the systolic murmur heard in the second, third, and fourth left interspaces in the later stages of such cases, is due to tricuspid regurgitation.

Before considering the arguments for and against these different theories, I may say that the question is still an open one, the balance of evidence, in my opinion, being in favour of the first or pulmonary view. I do not think, for the reasons to be presently given, that Dr Balfour has conclusively proved his point, on the contrary, there seem to me to be grave objections to his view, and I do not see that anything which has as yet been advanced, conclusively negatives the first or purely pulmonary theory.

Dr Balfour's Theory.

The facts and arguments on which Dr Balfour bases his theory, and the facts and arguments which seem to be opposed to it may be summarised as follows:—

1. (*For.*) That in organic diseases of the mitral valve a systolic, basic, and apparently pulmonary murmur is often present. Admitted that this is so.

2. (*For.*) That this supposed pulmonary murmur is, as Naunyn was the first to suggest, not pulmonary at all, but that it is due to mitral regurgitation, and is conducted to the second left interspace through the dilated appendix of the left auricle.

(*Against.*) While granting that, if the appendix of the left auricle were dilated and were in contact with the chest wall, the systolic murmur of mitral regurgitation would probably be conveyed to the second left interspace; and that, as Dr Balfour points out, Naunyn's theory has been accepted as highly probable by many leading Continental authorities, it must be allowed that it has not been accepted as conclusively proved, more especially by many competent British observers. But even granting that it is true for some cases of organic disease (*i.e.* granting that cases of mitral regurgitation do occur, in which the usual symptoms and signs of that condition, more especially a systolic murmur audible at the apex, are present, and in which the auricular appendix is dilated and is in contact with the chest wall), it by no means follows that the chlorotic murmur with which we are now more particularly concerned is produced in the same manner.

The arguments with which Naunyn supports his theory are these:—

(a) (*For.*) That the murmur has its point of maximum intensity at a point an inch and a half to the left of the sternum.

(*Against.*) Speaking for myself, I have in several cases of anæmia failed to satisfy myself that the position of the maximum intensity is so sharply defined as Dr Balfour's statements seem to indicate. The murmur has seemed to me in several cases quite as loud half an inch to the left of the

sternum as over the so-called auricular area, *i.e.* an inch and a half to the left of that bone. Hayden, who is admitted to be a trustworthy observer, goes much further in this respect than I am disposed to do, for he states that the murmur is best heard at midsternum.

But even granting that the point of maximum intensity of the murmur, in cases of chlorosis, is in the position which Dr Balfour describes, it does not seem to me by any means to prove that the murmur is not pulmonary. In the first place, Sibson has shown that 'in the large majority of cases' which he examined, 'the greater part (in 25 of 45 instances), or the whole (in 14 of 45 instances), of the artery, bore to the left of the sternum, and presented itself behind the upper costal cartilages and their spaces from the first cartilage to the third space;' ¹ and further, that the average breadth of the vessel in Pirogoff's five front views of the healthy heart was an inch and a quarter. ² I see nothing improbable, therefore, in a pulmonary murmur having (in many cases) its point of maximum intensity considerably to the left of the sternum.

In the second place, there seems good reason to suppose, as Dr Russell's observations seem to show, that in consequence of the dilated condition of the heart (of the right ventricle more especially) the relative position of the parts is somewhat altered, and that the pulmonary artery may be displaced forwards, and perhaps somewhat to the left of its usual position.

(*b*) (*For.*) That this point does not correspond to the origin of the pulmonary artery, but does exactly correspond to the spot where the appendix of the left auricle comes up from behind, just to the left of that artery, and that in cases of this description (*i.e.* where the murmur is loudly heard in the second left interspace) the appendix of the left auricle is dilated, and is closer to the chest and therefore nearer to the ear than usual.

(*Against.*) It is not proved that the appendix of the left auricle is dilated in cases of chlorosis (with which we are at present more immediately concerned). On the contrary there are, as we shall presently see, positive observations to the

¹ Russell Reynold's *System of Medicine*, vol. iv. p. 35.

² *Ib.* p. 115.

contrary; and there are, as Dr Russell has pointed out, grave anatomical objections to the theory that the left auricular appendix is in closer contact with the chest wall, in cases of chlorosis, than in health. This point will be again referred to.

But granting for the moment that cases of mitral regurgitation do occur, in which the left auricular appendix is dilated, and in closer contact to the chest wall than usual, and in which the systolic murmur produced at the mitral valve is heard as loudly in the second left interspace as at the apex, that is no reason for supposing that in a case of anæmia,—in which a murmur is heard in the second left interspace, and in which there is no murmur, be it observed, at the apex, and no other indication of mitral regurgitation present,—this apparently pulmonary murmur is of mitral origin.

The fact that the murmur is better heard over the auricular area than over the apex—the normal position of maximum intensity for mitral murmurs—Naunyn explains by attributing it to the better conduction of the murmur along the course of the regurgitating blood current, the fluid veins producing sonorous vibrations louder at the point of impingement than at that of origin, a view which is adopted and endorsed by Dr Balfour in support of his theory. He says, ‘the fluid veins formed in the early stage of chlorotic regurgitation are of low tension and but little force, hence the vibrations they originate are but slightly propagated to the left ventricle, and only with difficulty from it to the chest wall in the mitral area, where they are heard as an impure first sound. But on the other hand these vibrations are readily communicated to the wall of the auricle on which these fluid veins impinge, and are easily transmitted to the chest wall with which the auricular appendix is in contact, becoming audible in the auricular area as a distinct murmur.’¹

Against this view it may be argued that if the fluid veins produce sonorous vibrations louder at their point of impingement than at their point of origin in one case, why should they not do so in all? In other words, if this theory be correct, we ought to hear the systolic murmur of organic mitral

¹ *Diseases of the Heart*, p. 177.

regurgitation much more frequently and much more loudly in the second left interspace than is admitted by the majority of British observers, at all events.

Again, it may be asked if this explanation is correct in cases of chlorosis, and if, as Dr Balfour admits, 'the auricle, at the moment of ventricular systole, is not only full, but somewhat tense from unusual dilatation,' and if the fluid veins produce sonorous vibrations louder at their point of impingement than at their point of origin, why is it that the murmur is not heard in the back, under the left scapula, as the murmur of organic regurgitation so often is?

3. (*For.*) That in some cases of mitral disease, the apparently pulmonary, but in reality auricular murmur, is occasionally actually louder than that audible in the mitral area, and that the murmur of mitral regurgitation is sometimes only to be heard in the first named situation, *i.e.* in the second left interspace. Dr Balfour indeed goes so far as to state that 'a more or less distinct murmur in this (*i.e.* the auricular) area, is one of the earliest indications of mitral regurgitation from whatever cause.'¹

(*Against.*) While admitting that some authorities, Rosenstein for example, support this view, it is certainly contrary to the recorded opinion of almost all the best observers in this country, and I am not prepared to admit that a systolic murmur heard in the second left interspace is, *per se* (as Dr Balfour states in the following passage), indicative of mitral regurgitation.

'In chlorosis,' says Dr Balfour, 'in which all these phenomena, to which I may now comprehensively refer under the head of cardiac dilatation consecutive to spanæmia, exist only in the very slightest degree, this pulmonary or rather auricular murmur is always present, and is often—so often as to constitute it almost invariably—the sole sign of mitral regurgitation in these cases.'²

4. (*For.*) Dr Balfour claims 'that Naunyn's view is even more applicable in chlorosis than in any other form of heart affection, because the essential cardiac lesion in chlorosis

¹ *Diseases of the Heart*, second edition, p. 163. ² *Ibid.* p. 172.

is muscular relaxation and progressive dilatation, hence at a comparatively early stage of the disease the dilated right ventricle has separated the left ventricle from the chest wall, while the dilated appendix of the left auricle has been *pari passu* brought into closer contact with it.¹

‘The peculiar position of the murmur is due,’ says Dr Balfour, ‘to the altered position of the heart. ‘This is due to the dilatation of the right ventricle, which dilates *pari passu* with the left ventricle, and, like a water cushion, separates it from the chest wall, leaving the dilated appendix of the left auricle the only part of the left side of the heart in contact with the chest wall.’²

Dr Balfour further claims that the pulsation of the auricular appendix can be seen, felt, and graphically recorded by the cardiograph. He says—‘In this situation, the dilated appendix not infrequently gives rise to so distinct a pulsation that its movements can be traced by the cardiograph, and the history of several such cases has been published, and their cardiograms figured by my former resident, Dr George Gibson, while the pulsation is so well marked and forcible in some of these cases, that the late Dr Hughes Bennett sent me on one occasion a case of chlorosis as a case of aortic aneurism.’³

Against these statements it may be argued *firstly*, that Naunyn’s explanation seems more particularly to apply to those cases of mitral regurgitation in which a systolic murmur is audible *at the apex*, *i.e.* in the usual mitral area, *as well as* in the second left interspace; but that in the earlier stages of chlorosis, the apparently pulmonary murmur is, as Dr Balfour himself admits, usually the sole sign of mitral regurgitation; in other words, the usual evidence of mitral regurgitation, *i.e.*, a systolic murmur in the mitral area, is wanting.

Secondly, that in the later stages of anæmia (chlorosis) a true mitral murmur, *i.e.* a murmur audible at the apex—in the mitral area—does actually occur. This Dr Balfour himself admits, and he explains it in the following manner:—

¹ *Diseases of the Heart*, second edition, p. 176.

² *Edinburgh Medical Journal*, Oct. 1882, p. 295.

³ *Diseases of the Heart*, p. 175.

'By and by, as the regurgitation increases, and the ventricle hypertrophies, these fluid veins gain force sufficient to be communicated through the ventricle also, hence in the later stages of chlorosis we have a mitral murmur associated with the auricular one. It is however quite possible that this murmur in the mitral area is really tricuspid, due to the increased dilatation of the right ventricle, the apex of which may even occupy the mitral area; this not infrequently occurs in mitral stenosis, it is not an improbable event in chlorosis, and it is of little consequence which explanation we accept, the actual truth probably embracing both conclusions, being sometimes due to the one cause and sometimes to the other.'¹

Now I agree with Dr Balfour in thinking that in many cases of advanced chlorosis the systolic murmur, which is heard at the apex, is due to mitral regurgitation, but I differ from him in as much as I believe that it is a distinct murmur from that heard in the second left interspace; and I am unable to accept the theory which he advances to explain the supposed fact that a mitral murmur may, in the earlier stages of the condition be confined to the base, while in the later stages it is heard at the apex. For is it not the fact that as cases of chlorosis (and more especially of progressive pernicious anæmia, in which the same sequence of events occurs), advance, that the degeneration of the heart muscle increases, and that dilatation of the heart cavities, with increased feebleness of action, rather than hypertrophy with increased force of contraction, occurs? If this is so, Dr Balfour's explanation obviously cannot hold good; and he himself states that 'the essential cardiac lesion in chlorosis is muscular relaxation and progressive dilatation.'²

The order of recovery, too, seems to be opposed to this view. If the hypertrophy of the left ventricle is the cause of the mitral murmur being audible at the apex in advanced stages of the case, and if the mitral and so-called auricular murmurs depend on one and the same cause, *i.e.* upon mitral regurgitation, why, in cases which recover, should the basic murmur persist long after the apex murmur has disappeared?

¹ *Diseases of the Heart*, second edition, p. 177.

² *Ibid.* p. 176.

It can hardly be suggested that the left ventricle becomes weaker during the process of recovery; and if both murmurs depend on one and the same cause, *i.e.* upon mitral regurgitation, and if the ventricle does not become weaker, both murmurs, surely, ought to disappear at one and the same time.

Thirdly, Dr Russell has shown that in a case of progressive pernicious anæmia, in which both pulsation and bruit were present in the second left interspace during life, the auricular appendix was not dilated, and was not in contact with, but was far removed from, the chest wall, and that the pulsation, percussion-dulness and bruit were respectively seen and heard over the conus arteriosus.¹

Dr Balfour's counter argument against this, 'that the position of the auricle *post-mortem* is no proof of its state during life, and where there is no mitral stenosis, an auricular appendix beating in the second interspace during life, may very well empty itself and contract out of sight in the act of dying,'² is not, to my mind, a very convincing one. In the first place, our knowledge of the normal position of the appendix (including Naunyn's own observations, which Dr Balfour quotes as one of his main arguments), has been largely acquired by the same means which were adopted in this case. And in the second place, in cases of progressive pernicious anæmia, such as this was, the heart is usually (I think I may say invariably) relaxed and flaccid after death. It seems unlikely, therefore, that in this case the appendix emptied itself and contracted out of sight.

Fourthly, That when the right cavities of the heart are dilated, as Dr Balfour admits is the case, in *chlorosis*—the condition we are considering—the left auricular appendix is usually quite invisible from the front, a fact which Dr Russell has also urged. I have had several opportunities of verifying this statement during the past year, two of the cases being typical examples of pernicious anæmia. In none of these cases was the appendix much dilated, indeed in one of the cases of pernicious anæmia it was considerably smaller than usual. Dr Russell says—'It is further recognised that, in

¹ *Edinr. Medical Journal*, Nov. 1882, p. 408. ² *Ibid.* Sept. 1882, p. 197.

debility, owing to dilatation of the right ventricle, the left is displaced outwards and backwards; or a change occurs which may be regarded as a rotatory movement of the heart round its longitudinal axis; and this must be conceded as having a displacing effect on the auricle analogous to what it has on the ventricle of the same side.' Further, Dr Russell argues that, since 'the origin or root of the appendix is overlapped in part by the pulmonary artery, so to reach the parietes the appendix has to traverse a course equal to the diameter of that vessel. Any increase in the diameter of the artery, from increase of its contents will thus place the appendix deeper in the chest.'¹

I can from personal observation testify, as Dr Russell's argument implies, that the pulmonary artery is dilated in (some) cases of pernicious anæmia, presumably therefore it is dilated in some cases of chlorosis.

It must be at once conceded that pulsation is frequently to be observed in the second left interspace in cases of chlorosis. I cannot, however, say that I have ever been able to satisfy myself that it was auricular; and, like Dr Broadbent, I am not at all convinced that Dr Gibson's latter tracings, to which Dr Balfour particularly refers (see *Edinburgh Medical Journal*, October 1882, p. 294), prove the pulsation to be produced by regurgitation into the ventricle from the auricle. The tracings published by Dr Russell in the *British Medical Journal* of June 2, 1882, seem also opposed to Dr Gibson's view.

Further, in common with Dr Broadbent, Dr Goodhart, Dr Russell and other observers, whose pathological experience is not inconsiderable, I have not met with any case of cardiac dilatation—certainly not any case of anæmia, and I have had an opportunity of examining seven or eight cases, after death—in which the left auricular appendix was so markedly dilated 'as to warrant the belief that it could have been the cause of the extensive pulsation claimed for it by Dr Balfour in the second and third left spaces.'

Again, Dr Russell claims to have frequently satisfied himself that the pulsation in the second interspace in cases

¹ *Edinr. Med. Jour.*, Aug. 1882, p. 131. ² *Brit. Med. Jour.*, Aug. 26, 1882, p. 354.

of organic mitral disease, is due to the dilated right ventricle. I can corroborate Dr Russell's statement in this respect, inasmuch as I have in several cases of right-sided dilatation—notably in a case of pernicious anæmia—found that a needle passed into the second left interspace transfixes the conus arteriosus of the right ventricle, and did not transfix the pulmonary artery, as it does under normal circumstances.

To the other points previously advanced in support of and against Dr Balfour's theory, the following must be added:—

5. (*For.*) That the pulmonary second sound is accentuated.

We all, of course, know, that in mitral regurgitation, the pulmonary second sound is accentuated in consequence of the increased blood-pressure in the pulmonary artery, which results from the mitral lesion. If, therefore, the accentuation occurs in the earlier stages of chlorosis, and if it cannot be satisfactorily accounted for in any other manner (than as the result of mitral regurgitation), we must of course admit that it is strongly corroborative of Dr Balfour's theory, always premising that there is no fatal objection to that view.

(*Against.*) Now, I at once admit that, in the later stages of anæmia, the pulmonary second sound is often intensified, and that the accentuation may be due to the mitral regurgitation which is often present in the advanced stages of that condition. In the earlier stages of chlorosis, too, I have frequently (though not invariably) found the pulmonary second sound louder than the aortic. I have been in the habit of regarding the accentuation, which occurs in the early stages of chlorosis, as apparent rather than real; in other words, I have supposed that the aortic second sound was diminished in intensity, while the pulmonary retained its normal loudness. Possibly I may have been mistaken in this view, for the fact that the blood pressure in the peripheral arteries is increased rather than diminished in anæmia, would probably lead to accentuation rather than diminution of the aortic second sound;¹ and

¹ It is well established that the blood-pressure is increased in the earlier stages of anæmia. The increase is, I believe, due to two causes viz., (*a*) increased adhesions of the red corpuscles to the capillary walls, (*b*) contraction of the peripheral blood vessels, the result of anæmia of the vaso-motor centre.

I am certainly not prepared to say that true accentuation of the pulmonary second sound does not occur, even in the earlier stages of chlorosis.

But even granting that the accentuation were always present and well marked, I cannot admit that it is sufficiently strong evidence to counterbalance the grave objections which have been advanced against the mitral regurgitation view. In particular, the objections which Dr Russell has so ably urged, and which I can not only corroborate but also strengthen, viz.:—(1) that in the most advanced stages of anæmia, as seen after death, the left auricle appendix is not dilated (in one of my cases it was actually smaller than normal); and (2) that when the right heart becomes dilated, as it does in conditions of anæmia, the appendix of the left auricle recedes from, rather than comes in closer contact with the chest-wall—seem to me to negative Dr Balfour's view. I feel obliged, therefore, to suppose, that if the pulmonary second sound is actually intensified, the accentuation must be due to some other cause than mitral regurgitation. One cause of the accentuation is, I believe, the diminished suction power which the left ventricle exerts in consequence of the relaxed and feeble condition of its muscular wall; the flow of blood from the lungs to the left heart is not facilitated (on the occurrence of the ventricular diastole) as it is in health. Another cause may possibly be found in the altered composition of the blood, though this is, to say the least, extremely doubtful. Dr Gaskell, indeed, whom I consulted on this matter, is inclined to think that a diminished number of blood corpuscles would facilitate rather than retard the flow of blood through the lungs; he writes—‘As to your question about the passage of the blood through the lungs in anæmia, I should think that probably the blood would pass through more easily for one reason at all events; the experiments of Ewald (*Archiv. f. Anat. u. Physiologie* 1877, ueber die Transpiration des Blutes) have shown that defibrinated blood passes through fine capillary glass tubes much more easily when there are few or no corpuscles in it than in the normal condition, so, for this reason alone, the blood-flow should be more easy. On the other hand, the

anæmic condition of the blood would probably excite the vasomotor centre, and so tend to constrict the blood vessels of different vascular areas, and therefore increase arterial pressure. There is, however, no necessity that the pulmonary vessels should take part in such constriction, and, indeed, the evidence points rather the other way, for excitation of the vasomotor centre, either directly or reflexly, by means of the stimulation of a sensory nerve, does not appear to diminish but rather to increase the flow of blood through the lungs, and similarly, I should imagine, that in the case of any excitation of the vasomotor centre, through an anæmic condition of the blood, no constriction of the pulmonary vessels would take place, but if anything dilation. For both reasons, therefore, it is more likely than not that the passage of the blood through the lung vessels takes place with greater facility when there are fewer red blood corpuscles.'

Dr Russell's Theory.

Let us now turn to Dr Russell's theory. He believes:—

(1) That the murmur heard in the second left interspace, in the earlier stages of chlorosis, is generated in the pulmonary artery.

(2) That the murmur is due to a relative constriction of the vessel (*i.e.* of the pulmonary artery), produced by the pressure of the dilated left auricle, which is situated behind it.

Now this theory necessarily presupposes that, during the ventricular systole, the pressure of the blood in the left auricle is greater than the pressure of the blood in the pulmonary artery; for otherwise the pulmonary artery could not, of course, be constricted by the pressure of the auricle. That this is so Dr Russell maintains; and he contends that the excessive pressure (if I may so term it) in the left auricle is due to a regurgitant current being propelled into it (the left auricle), by the left ventricle through the mitral orifice.

The explanation which he gives is as follows:—'Owing to the tension in the pulmonary vessels, the fulness of the auricle must at all times be increased; in fact, the blood

coming from the lungs pours into it as rapidly as it empties itself into the ventricle, the result being that its cavity, although enlarged by sharing in the debilitating influences in existence, is already full before the ventricular systole not only prevents further relief to the tension in the pulmonary circuit, but throws back upon it (the auricle) the blood embraced by the segments of the mitral valve as they swing to close the auriculo-ventricular orifice, as well as the column of regurgitant blood from the ventricle.¹ And again, 'The question, then, arises as to what force may act *through* the left auricle, and is it greater than that of the right ventricle? Take mitral regurgitation: there a stream, at times of considerable magnitude, passes back into the auricle with all the force exerted by the systole of the left ventricle; and, as we cannot assume the left auricle to be a vacuum ready to receive this regurgitating stream, there must be a backward flow through it, nearly as much greater in force than the flow in the pulmonary artery as the systole of the left ventricle is greater than the right. . . . The next question to consider is, whether the tension in the left auricle is greater than that in the pulmonary artery at the moment of ventricular systole.'² . . . 'When it is remembered that there is not only a column of blood sent with the force of the left ventricle into this tense auricle, but also that the mitral cusps, instead of being held well down into the ventricle and presenting a concavity towards the auricle, so as thereby to relieve the auricle and 'make room for the returning blood without hindrance,' must, on the contrary, owing to the enfeebled state of the papillary muscles, be allowed greater latitude of movement towards the auricle, it must be granted that the auricular tension bears a fixed relation to the strength of the left ventricle, and is therefore greater than that in the pulmonary artery.'³

In opposition to this theory Dr Balfour maintains that 'it is absolutely impossible that the left auricle can ever so compress the pulmonary artery;' . . . and 'that it is obviously impossible that the circulation could be carried on under these

¹ *Edinburgh Medical Journal*, Aug. 1882, p. 134.

² *Ibid.* Nov. 1882, p. 411. ³ *Ibid.* Nov. 1882, p. 412.

conditions.' It is unnecessary, I think, to detail the facts and arguments with which Dr Balfour supports these objections, and the facts and arguments which Dr Russell urges in reply, for (granting for the moment that Dr Balfour's objections are invalid) I maintain :—

That even if such excessive intra-auricular pressure could occur, it certainly is not present in the early stages of chlorosis—the condition which we are now considering. Such excessive intra-auricular pressure could only be produced by extremely free mitral regurgitation. In all cases of mitral regurgitation there is probably a considerable direct obstacle (both valvular and muscular), in addition to the blood pressure in the auricle, opposed to the force of the left ventricle ; while there is no direct obstacle, but only the blood-pressure in the pulmonary artery, opposed to the force of the right ventricle. We cannot, therefore, with fairness say, because the left ventricle is much stronger than the right, it will in mitral regurgitation raise the blood pressure in the left auricle higher than the right ventricle will raise the blood pressure in the pulmonary artery. The right ventricle, though much weaker, is acting (more especially when the tricuspid is sound) at an immense advantage, and undoubtedly propels a much larger quantity of blood into the pulmonary artery than the left ventricle propels (in any ordinary case of mitral regurgitation) into the left auricle.¹

Now, there is not sufficient evidence, I maintain, to prove that any regurgitation through the mitral valve occurs in the early stages of chlorosis and anæmia, much less the free regurgitation, which would be required to produce the excessive pressure in the left auricle, which Dr Russell's theory requires.

The only evidence which Dr Russell produces in favour of mitral regurgitation in the early stages of chlorosis, is an accented pulmonary second sound. 'It will not be seriously denied here,' says Dr Russell, 'that in these affections there is a relative insufficiency of the mitral valve, the result of a

¹ The reader is referred to the original papers. See the *Edinburgh Medical Journal*, August and September 1882, p. 198 ; and November 1882, p. 409.

debilitated and relaxed condition of the cardiac muscle, including the muscoli papillares. This insufficiency, however, is not always evidenced by a bruit at the apex, the point at which mitral bruits are ordinarily audible; but regurgitation is assumed¹ from the accentuation of the pulmonary second sound, and from the presence of a systolic murmur in the pulmonary area.²

I have already given, in detail, the facts and arguments which are, I consider, opposed to Dr Balfour's view, and have previously stated that the presence of a systolic murmur in the second left interspace is not, in my opinion, *per se* (I may add, nor when conjoined with an accentuated pulmonary second sound) sufficient evidence of mitral regurgitation. And Dr Russell himself maintains that this systolic murmur is in reality produced in the pulmonary artery. According to his own showing, then, the only evidence of mitral regurgitation which remains is accentuation of the pulmonary second sound. This I maintain is insufficient evidence.

Since this lecture was written, Dr Russell seems to have modified the view which he originally proposed, inasmuch as he no longer insists that the increased tension of the left auricle (which his theory necessarily supposes is present during the first part of the ventricular systole) is due to mitral regurgitation. In the passage quoted in the foot-note, from his instructive paper in the *British Medical Journal* of June 2d 1883, he suggests that the increased tension of the left auricle may be due to the incomplete emptying of the left ventricle during its systole, and consequent imperfect relief to the full auricle.³

But if this view be correct, should we not expect to hear a

¹ Dr Russell is here speaking of Dr Balfour's theory; he himself believes that the systolic basic murmur is not mitral, but pulmonary,

² *Edinburgh Medical Journal*, Aug. 1882, p. 130.

³ 'I shall now endeavour to explain the clinical phenomena. The accentuation of the pulmonary second sound, if no lung affection be present, must be taken as indicating an abnormal accentuation of blood behind the mitral orifice. Whether this be due to the incomplete emptying of the left ventricle during its weakened systole, and consequent imperfect relief to the full auricle, or, from the first, due to a certain amount of regurgitation, or to both these causes, it is unnecessary to

systolic pulmonary murmur as a necessary accompaniment of all cases of organic mitral disease, in which the pulmonary second sound is accentuated? We can hardly suppose that the distention of the left auricle is great in the early stages of chlorosis; and if a small amount of distention is sufficient to constrict the pulmonary artery, and to produce a systolic murmur, should we not *a fortiori* expect to have a systolic pulmonary murmur produced in those cases of mitral stenosis, for example, in which we may legitimately conclude that the distention of the auricle is still more considerable? It may, of course, be argued (1) that a systolic murmur is sometimes heard in the pulmonary area in cases of mitral stenosis; and (2) that in those cases of mitral stenosis in which a systolic pulmonary murmur is not present, the left auricle is not dilated. In support of the latter proposition, it may be urged that in mitral stenosis the cardiac muscle is not degenerated, as it is in the case of anæmia, and that the left auricle, for a time at all events, is able to resist the excessive blood-pressure in its interior, and does not dilate. But looking at the matter from the broad ground of clinical experience, most observers will, I think, agree that cases (such as mitral stenosis, mitral regurgitation, fatty heart, etc.), are frequently met with, in which the left auricle is quite as much distended as we can legitimately suppose it to be in the earlier stages of chlorosis, and in which there is no pulmonary systolic murmur. If this general proposition be granted, we must of course conclude that the pulmonary murmur, met with in the earlier stages of chlorosis, is not produced by the pressure of the distended left auricle upon the pulmonary artery, but that it is due (either wholly or in part) to some other condition or conditions.

I feel obliged, therefore, to dissent from Dr Russell's theory; and there are (as I have already pointed out in discuss here. The fact of accumulation of blood in the pulmonary circuit, including the left auricle, is sufficient for our present purpose, and is warranted by the evidence given by pulsation at the root of the neck, by the course of the external jugulars becoming visible, and perhaps by pulsation appearing over the right ventricle, that a like accumulation is taking place in the right chambers of the heart and the large vessels leading to it.'

detail), in my opinion, grave objections to Dr Balfour's view. I am compelled, therefore, by the method of exclusion, to fall back upon the purely pulmonary theory, and to ask whether there is any conclusive reason why the murmur should not be generated in the pulmonary artery itself, irrespective of any constriction by the pressure of the auricle, such as Dr Russell's theory implies.¹

The Purely Pulmonary Theory.

Dr Balfour argues that the murmur cannot be pulmonary:—

(1.) Because 'there are—in chlorosis—no causes of murmur operative at the pulmonary orifice which are not at least as active at the aortic opening, so that a pulmonary murmur would certainly be accompanied by an aortic murmur also, and the latter would, of course, be propagated along the course of the aorta, and more or less distinctly into the carotids.'² This argument does not, however, appear to be conclusive. In the first place, we might as well say, in opposition to Dr Balfour's own view—the auricular theory—that the murmur cannot be mitral, because there are no causes of murmur operative at the mitral orifice, which are not at least as active at the tricuspid opening, so that a mitral murmur would certainly be accompanied by a tricuspid murmur; and since it is generally admitted, and as Dr Balfour himself allows in the passage quoted below, that the tricuspid murmur is of later occurrence than the mitral murmur, Dr Balfour's auricular theory falls to the ground. Dr Balfour says, 'shortly after the appearance of the primary hæmic murmur, a tricuspid murmur and jugular undulation are found to be developed. This is naturally accompanied by a

¹ There seems to me to be no sufficient evidence to justify the belief, that the murmur heard in the second left interspace, *in the early stages of anæmia*, is due to tricuspid regurgitation, as Parrot supposed. The mere presence of a murmur in that position, in the absence of the usual signs of tricuspid regurgitation, is insufficient evidence to justify such a belief. It is, however, quite possible that, in the later stages of anæmia, a tricuspid murmur may be heard in this situation, as Dr Russell supposes.

² *Edinburgh Medical Journal*, Oct. 1882, p. 294.

pulmonary and also by an aortic systolic murmur the active cause in the production of both these murmurs being the large blood-waves sent on by the dilated and hypertrophied ventricles, as was first, I believe, pointed out by Beau.¹

In the second place, I am not prepared to admit, unconditionally, that there are no causes of murmur operative at the pulmonary orifice, which are not at least as active at the aortic orifice. It appears to me quite possible that such causes may exist in the respective conditions of the two ventricles; in the respective resistances which the arterial blood meets with at the orifice of the aorta and in the systemic circulation, and which the venous blood meets with at the orifice of the pulmonary artery and in its passage through the lungs; and in the respective physical conditions (calibre, thickness of coat, relationship to the chest wall, etc.) of the aorta and pulmonary artery.

In the third place, I believe that aortic murmurs are sometimes present in the early stages of chlorosis, possibly they would be more frequently audible in such conditions, if it were not for the fact, that they are so soft and faint as to be obscured at the base by the pulmonary murmur, and of such low tension, and of such little force, as to be inaudible over the course of the aorta and in the carotids.

(2.) Because the point of maximum intensity of the murmur 'is not over the pulmonary artery at all, but from one to two inches to the left of the sternum, in the second interspace.'²

This argument also fails to convince me, for the reasons already detailed. (See p. 190.)

(3.) Because no murmur of strictly pulmonary origin could possibly be referred to all four orifices in turn, as has been the case with the hæmic murmur; and second, because however singular a murmur of mitral regurgitation in this position may seem to be, its causation is by no means difficult to understand.³

Neither of these reasons seems to me to exclude the pulmonary hypothesis. The mitral origin of the murmur,

¹ *Edinburgh Medical Journal*, Oct. 1882, p. 295. ² *Ibid.* p. 294.

³ *Diseases of the Heart*, second edition, p. 173.

which Dr Balfour supports, would be as effectually excluded by the first reason, as he argues the pulmonary origin is; for no murmur of strictly mitral origin could possibly be referred to all four orifices in turn. The only legitimate conclusion to be drawn from the first reason is, that in chlorosis, murmurs may be generated at more than one orifice, a conclusion which we all allow. The second reason, even if admitted, does not exclude the pulmonary hypothesis, but only shows that a mitral murmur can be heard in the neighbourhood of the pulmonary artery.

I feel compelled, therefore, to differ from Dr Balfour; for I do not see that any argument which has been as yet advanced conclusively negatives the purely pulmonary theory.

As I have previously stated, the *sudden* propulsion of a *large* blood-wave, of *abnormal* (spanæmic) composition into the vessel, which is possibly dilated, seems to me an efficient cause for the production of the murmur; and we know, as a matter of fact, that in cases of chlorosis these conditions are actually present—the chlorotic heart is unusually irritable and contracts with unusual suddenness; even in the earlier stages, there is some dilatation of the right ventricle; the blood is spanæmic, and in some advanced cases, *i.e.* fatal cases of pernicious anæmia, the pulmonary artery is dilated.

Dr Balfour himself states, in the passage I have quoted above, that aortic and pulmonary murmurs do occur in the later stages of chlorosis, and that the active cause in the production of both is the large blood-wave sent on by the dilated and hypertrophied ventricles. Now, if aortic and pulmonary murmurs can be produced by this cause in the later stages of chlorosis, and after, as Dr Balfour argues, tricuspid and mitral regurgitation have occurred; should they not *a fortiori* be produced in the earlier stages, before, as I maintain, there is sufficient evidence of mitral and tricuspid regurgitation, for would not the presence of a leak at the tricuspid and mitral orifices diminish the size of the blood-wave, and so, other things being equal, be likely to interfere with the production of, rather than to cause such murmurs?

The Differential Diagnosis of Cardiac Murmurs.

Given the presence of a murmur over the præcordia, we have to determine :—

1. Whether it is exocardial or endocardial.
2. If exocardial, whether it is a pleural, a pericardial, or a pericardial-pleural murmur.

If endocardial :—

3. The valve at which it is produced, and whether it is direct or regurgitant.
4. Whether it is organic or functional.
5. If organic, the extent and gravity of the lesion.

Many of these points I shall afterwards have to consider in detail, in treating of the diagnosis and prognosis of the individual cardiac affections, but it may perhaps be well, even at the risk of some future repetition, to consider the subject now as a whole, and to point out the leading facts and circumstances which enable us to form an opinion on these important questions.

Step No. 1. Differential Diagnosis of Exocardial and Endocardial Murmurs.

This question is, as a rule, easily determined by attention to the following points :—

1. *The Rhythm of the Murmur.*—Pleural friction sounds are of course at once distinguished (except in the case of the pericardial-pleural friction murmur, which I shall presently refer to) from cardiac murmurs by the fact, that they correspond in rhythm or frequency to inspiration and expiration, and that they do not correspond to the pulsations of the heart.

Exocardial murmurs (pericardial and pericardial-pleural friction murmurs) correspond in rhythm or frequency to the pulsations of the heart, but their synchronism is, as a rule, much less perfect, *i.e.* they do not correspond so exactly to the heart sounds, as do endo-cardial murmurs.

Pericardial murmurs, when typical, are double (to-and-fro friction sounds), but the murmur of combined aortic stenosis and incompetence is also double; hence in the majority of cases the problem resolves itself into the differential diagnosis of these two conditions, *viz.*, pericarditis and double

aortic disease—a point which will afterwards be considered in detail.

The rhythm of exocardial murmurs is, as a rule, more variable than the rhythm of endocardial murmurs. The character and the rhythm of the murmur is apt to change from day to day, from hour to hour, or even during the actual examination of the patient, a fact which is explained by the circumstance that the relative positions of the two opposed and roughened surfaces of the pericardium may be materially modified by alterations in the position of the patient, and the pressure of the stethoscope.¹ The fact that alterations in tone and rhythm can be produced by the pressure of the stethoscope is highly characteristic of pericardial murmurs.

2. *The sound characters of the murmur.*—Exocardial murmurs are friction sounds, and, as a rule, have a harsh, grating character; they usually appear to be superficial. But this and their other characters I have previously described. (See p. 167.)

3. *The point of maximum intensity of the murmur, and the direction in which it is propagated.*—Exocardial murmurs are, as a rule, best heard over the centre of the right ventricle or at the base of the heart; but they have no special points of differential maximum intensity as endocardial murmurs have. Exocardial murmurs are often only heard over a very limited area, and they are not propagated in any definite direction as endocardial murmurs are. (See p. 182.)

4. *The associated symptoms and history of the case.*—Exocardial murmurs appear abruptly, so to speak, and generally in the course of some constitutional affection such as rheumatic fever, Bright's disease, etc. Endocardial murmurs may appear abruptly and in the course of rheumatic fever,

¹ Endocardial murmurs are sometimes only audible in one particular position. The presystolic murmurs, for example, may disappear when the patient sits up, as Professor Sydney Ringer and others have noted. Again, other endocardial murmurs are very decidedly intensified by sitting up, walking, etc. But in these cases the rhythm of the murmur remains the same, and is not modified by alterations in position.

but, in many cases, the lesion which produces them develops very slowly. The absence of constitutional disturbance, and, especially, the fact (if such a history can be obtained) that the murmur is an old one, are strongly in favour of its endocardial origin.

Step No. 2. The Murmur is Exocardial; is it a Pleural, Pericardial, or Pericardial-pleural Murmur?

Ordinary pleural friction is at once distinguished by the fact, that its rhythm corresponds to the rhythm of the respiratory movements.

Pericardial-pleural friction is extremely rare. It occurs, as I have previously explained, when that portion of the pleura, which is reflected over the pericardium is inflamed; and it is produced by the movements of the heart rubbing this rough and inflamed portion of the pleura against the anterior wall of the chest, or against the visceral pleura which is in contact with it. The points by which we are enabled to distinguish pericardial-pleural friction from ordinary pleural friction are as follows —

1. Pericardial-pleural friction is generally best heard over the borders of the heart, *i.e.* where the visceral and parietal portions of the pleura come into contact; whereas ordinary pericardial friction is, as a rule, best heard over the centre of the cardiac dulness, *i.e.* over the centre of the anterior surface of the right ventricle.

2. Pericardial-pleural friction is, as a rule, more affected by the respiratory movements than ordinary pericardial friction. I have, in two cases, observed that it was decidedly increased by a full inspiration, *i.e.* when a larger portion of the two inflamed surfaces of the pleura were brought in contact. Walshe states that it is, as a rule, increased during expiration; and in such cases the murmur is probably produced by friction between the outside of the pericardium and the inside of the chest wall. When the patient takes a deep inspiration, the pericardial-pleural friction is sometimes replaced by ordinary pleural friction. This is not pathognomonic, for pericarditis is not unfrequently accompanied by pleurisy, and, in such cases, the pericardial friction may only

be observed over the præcordial region during a deep inspiration, and may then replace the pericardial friction sound.

3. In pericardial-pleural friction we should expect to find the symptoms of pleurisy, but there would be no indications of pericarditis; and *vice versâ*. It must not, however, be forgotten that many cases of dry pericarditis are unattended by any symptoms or signs except the to-and-fro friction murmur.

4. The pericardial-pleural friction murmur is even more variable than the ordinary pericardial friction murmur; and may even cease with certain pulsations of the heart.

Step No. 3.—The Murmur is Endocardial; at which valve is it produced, and is it direct or regurgitant?

The valve at which the murmur is produced is determined by observing:—

1. The point of differential maximum intensity of the murmur. (See page 180.)

2. The direction in which it is propagated. (See page 182.)

3. The effect which the lesion has produced upon the heart and circulation. It will be more convenient to consider this point when speaking of the character of the lesion. (See page 214.)

4. The relative frequency of the different valvular lesions. This is not a point of very much importance, for it is generally easy to decide the question by the points already mentioned (1, 2, and 3); but in some doubtful cases, as, for instance, in the case of a murmur heard over the base of the heart, the fact that the pulmonary orifice is very rarely diseased after birth, would, supposing that we could exclude inorganic conditions, be strongly in favour of the aortic origin of the murmur.

The question whether the murmur is direct or regurgitant is, of course, easily decided, by observing its rhythm. (See page 172.) When it is difficult or impossible to determine the rhythm, the sound characters of the murmur, and the effects of the lesion on the heart and circulation are the points which must be chiefly relied upon.

Step No. 4.—The Murmur is Endocardial; is it Organic or Functional?

It is sometimes very difficult, or even impossible, to give a positive answer to this question; in other cases it is decided with the greatest ease. The difficulty is greatest in the case of mitral systolic murmurs, which result so frequently from 'muscular' and 'relative' incompetence, and in which therefore we have to decide whether the muscular weakness of the heart is curable or not. In such cases it is only by taking a broad and general view of all the features of the case—independently of the mere physical examination of the heart itself—that a correct conclusion can be arrived at.

In practice, the first step in the differential diagnosis of organic and functional murmurs is *to determine the rhythm of the murmur*. The so-called functional murmurs are, as we have already seen, always systolic, and may be heard either in the pulmonary, aortic, mitral, or tricuspid areas. If, then, the murmur is diastolic or presystolic, we may with certainty conclude that it is organic. (As I have previously stated, Professor Austin Flint believes that a mitral lesion is not essential for the production of a presystolic murmur. The matter is perhaps of less practical moment than would at first sight appear. For since Professor Flint only claims to have noted this presystolic murmur, without mitral lesion, in cases of aortic incompetence; and since aortic incompetence is always an organic and serious lesion, the matter is, so far as our present purpose is concerned, comparatively unimportant, *i.e.* so far as concerns the decision whether the cardiac lesion is temporary and curable, or organic and incurable.)

If the rhythm of the murmur does not decide the question—that is to say, if the murmur is systolic—we must seek to determine the question by attention to the following points:—

(a.) *The presence or absence of the causes of inorganic murmurs on the one hand, or of organic murmurs on the other.*

The structural changes of a temporary kind which affect the heart muscle in anæmia and the continued fevers, are the great causes of the so-called inorganic or functional

murmurs. A murmur, then, occurring in a patient, who is neither anæmic nor suffering from one of the continued fevers, is probably organic. The reverse proposition (that murmurs, occurring in persons who are anæmic or suffering from one of the continued fevers, are functional) does not necessarily hold good. This caution more particularly applies to cases of rheumatic fever in which there is a strong tendency to inflammatory affections of the heart, but in which functional murmurs due to anæmia and to temporary and curable alterations in the heart muscle, are also frequently developed. Murmurs, then, which develop in the course of rheumatic fever, are probably, but not necessarily, organic. It is unnecessary to detail here the ordinary symptoms of anæmia; the pallor of the mucous membranes, and the presence of a venous hum in the neck, are the points of most importance.

(b.) *The condition of the heart and circulation.* This sometimes gives us important information, as in the following case:—

Case.—J. B., æt. 51, was admitted to the Newcastle-on-Tyne Infirmary under my care on 29th November 1878, complaining of shortness of breath and of swelling of the feet. He was very anæmic; a well-marked systolic murmur was audible over the aortic area; the left ventricle was not markedly hypertrophied. The pulse was slow and deliberate, and the apex of the sphygmographic tracing rounded. These characters which are seen in figure 55, seemed to show that the aortic murmur was not merely hæmic, but that it was due to organic stenosis of the aortic orifice.



FIG. 55.—Sphygmogram from the case of J. B., referred to in the text.

It must, however, be remembered, that free mitral regurgitation may be due to temporary and curable conditions; and that in such cases all the symptoms and signs of regurgitation due to organic and incurable disease (viz., accentuation of the pulmonary second sound, changes in

the right heart, shortness of breath, dropsy, etc.), may be present.

(c.) *The effect of treatment.*—This is a most valuable, and indeed, in some cases, the only certain means of deciding whether a murmur is functional or organic.

(d.) *The point of maximum intensity of the murmur and the extent of its propagation.*—These points may also give us some information. Anæmic murmurs are generally basic, and are most commonly heard over the pulmonary area, often also over the aorta, more rarely at the apex. Inorganic murmurs are not so well propagated as organic ones; aortic murmurs, therefore, which are carried into the vessels of the neck, and mitral murmurs, which are audible at the inferior angle of the left scapula, are probably organic.

(e.) *The history of the case.*—This may be of importance; a history of rheumatic fever would, other things being equal (*i.e.* in a doubtful case), be strongly in favour of the organic nature of the case.

The differential diagnosis of functional and organic murmurs will be considered in further detail when the individual valvular lesions are treated of. (See chapter V.)

Step No. 5.—The Murmur is Organic; what is the extent and gravity of the Lesion?

The extent of the lesion.—Other things being equal, the more extensive the lesion, the more serious the case. Now, in recent cases it may be very difficult, or even impossible to determine the exact extent of the lesion. It may be impossible, for instance, to say what proportion of the symptoms, is due to temporary conditions, and how far the morbid conditions may be restored or compensated.

In chronic cases the extent of the lesion is more easily determined, and in order to arrive at a correct conclusion the following circumstances must be taken into account:—

1. *The effect of the lesion upon the heart and the circulation.*—As we shall afterwards see, the great effect of all valvular lesions is to prevent the steady onward passage of the blood current. In seeking, therefore, to estimate the extent and

gravity of any valvular lesion, it is necessary to examine the effects produced :—(A.) In the cavities of the heart, and in the parts of the circulation which are situated *behind* the lesion—the *backward effects*; (B.) In the cavities of the heart and parts of the circulation which are situated *in front* of the lesion—the *forward effects*, as they may be termed. The more marked these effects, and the shorter the period which has been required for their full development, in other words, the more rapid the progress of the case, the worse the prognosis. It will be necessary, therefore, in the next place, to consider the effects which the different valvular lesions *tend* to produce on the circulation in front and behind, and the symptoms which result therefrom. I say, *tend* to produce, for it is essential to remember that serious valvular lesions may, for a time at least, be unattended by any apparent external effects or symptoms—a point which I have already insisted upon in speaking of the important ‘principle of compensation.’ On the other hand it is no less important to remember, that in these cases (*i.e.* in cases of serious valvular lesions, which are so perfectly compensated, as to be unattended by any apparent external effects or symptoms so long as the circulation is not, so to speak, put upon the stretch) there are internal effects (such as hypertrophy of the heart, etc.) which can be detected by physical examination.

The effects which the different valvular lesions *tend* to produce on the heart and circulation are as follows :—

MITRAL STENOSIS.

A. Backward Effects. (a) Blood stagnates in the left auricle, which becomes over-distended, hypertrophied, and dilated; (b) The pulmonary veins become engorged; (c) The lungs become congested: *lung symptoms*, consisting of shortness of breath, especially on exertion, going up stairs or up a hill; a tendency to catarrhal affections of the lungs and bronchi (especially chronic bronchitis), to œdema of the lungs, to hæmoptysis, to hydrothorax, etc., are apt to arise; (d) The increased blood-pressure in the pulmonary artery produces accentuation of the pulmonary second sound; and there is,

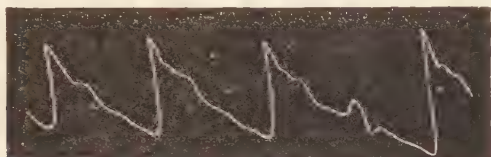
in many cases, reduplication of the second sound of the heart ; (e) The right ventricle becomes hypertrophied and dilated ; (f) Tricuspid regurgitation not unfrequently occurs with pulsation in the veins of the neck.¹ (The enlargement of the right cavities of the heart, both ventricle and auricle, and the presence of tricuspid regurgitation, can be ascertained by physical examination) ; (g) The systemic venous circulation is impeded, and the symptoms which result from this venous engorgement are often the first which attract the attention of the patient.² The peripheral parts, more especially the lips, nose, and ears, tend to become cyanotic, and the face somewhat full and swollen. Dropsy, commencing in the feet (increased by standing and walking, and therefore worse at night), gradually extends upwards, and finally involves the serous cavities as well as the subcutaneous tissues of the lower parts of the body. Engorgement of the portal vessels produces congestion of the liver, stomach, and hæmorrhoidal veins, with resulting enlargement of the liver, muddiness of the complexion or slight jaundice, dyspepsia, and piles. Congestion of the renal veins is attended with scanty and high coloured urine, which is loaded with urates, and often contains albumen. Interference with the return current from the brain may be attended with drowsiness and other indications of mental obfuscation.

B. Forward Effects.—Small and variable quantities of blood are passed into the left ventricle through the stenosed orifice ; consequently small and variable quantities of blood are pumped into the arterial system, the pulse being, therefore, small, unequal in volume, and irregular in time. (So long as the compensation is well maintained, the volume and rhythm of the pulse may not be much altered.) The occurrence of a second and imperfect ventricular contraction

¹ For the reasons previously given, the accentuation of the pulmonary second sound may diminish or disappear with the occurrence of tricuspid regurgitation.

² In this description I have sketched the backward effects in their anatomical, rather than their chronological sequence, venous engorgement and its resulting symptoms occurring long before many of the other conditions, such as tricuspid regurgitation.

in the sphygmographic tracing, such as is shown in fig. 56, is often observed.



Pressure $3\frac{1}{2}$ oz.

FIG. 56.—*Irregularity of the Pulse.*—W. M., æt. 50, admitted to Newcastle Infirmary 30th November 1878, suffering from the usual symptoms of mitral disease. The heart's action was extremely irregular. The left ventricle much hypertrophied. There was no rheumatic history. The symptoms were of two months' duration.

I have attempted in figs. 57, 58 to represent in a diagrammatic manner, the backward effects which mitral lesions produce on the heart and circulation.

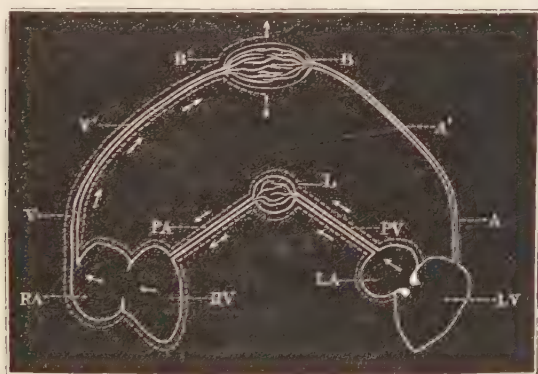


FIG. 57.—Representation of the effects of a lesion of the mitral valve (mitral stenosis in this case) on the heart and circulation. The effects of mitral regurgitation are the same, but there is, in addition, hypertrophy of the left ventricle. LV=left ventricle; LA=left auricle; PV=pulmonary veins; L=lungs; PA=pulmonary artery; RV=right ventricle; RA=right auricle; V=venæ cavæ; V'=venous system; B'=termination of the venous system in the systemic capillaries; A=the aorta; A'=the arterial system. The arrows show the direction of the backward pressure. The dotted lines show the effects of the obstruction (dilatation) on the different parts.

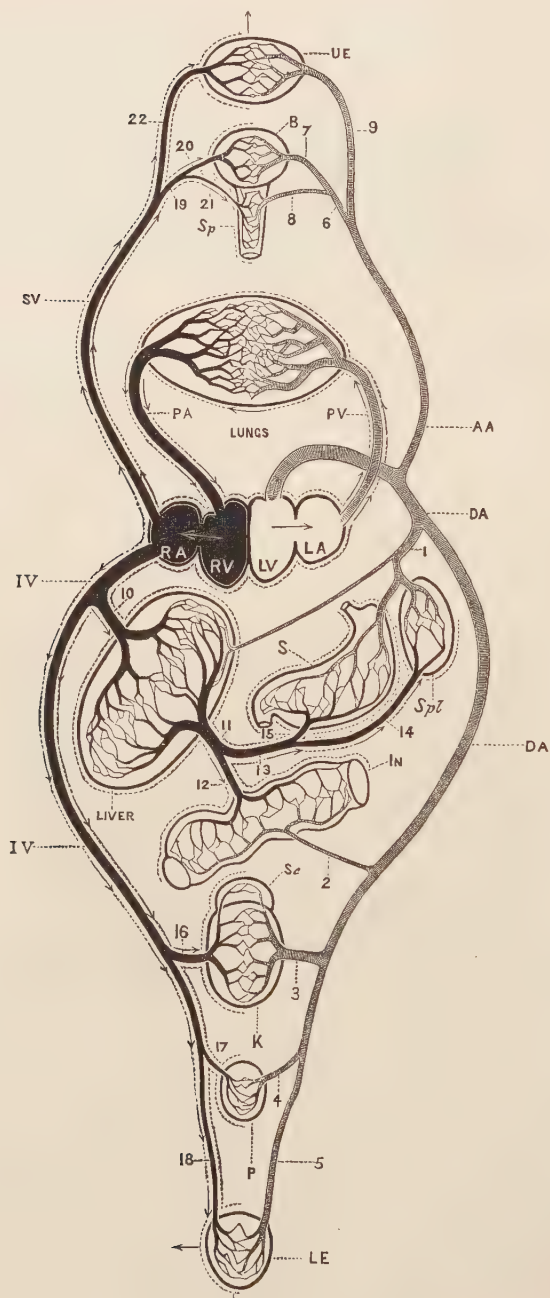


FIG. 58.

MITRAL REGURGITATION.

A. *Backward Effects*.—The backward effects are similar to those which are produced by mitral stenosis. As a rule grave disturbances of the circulation are manifested earlier in mitral regurgitation than in stenosis—a fact which is easily understood when we remember that in many cases the former results from degeneration and failure of the heart muscle, independently of any lesion of the valvular segments.

B. *Forward Effects*.—The pulse is small, and in advanced cases (*i.e.* after the failure of compensation), it is usually irregular.

AORTIC STENOSIS.

A. *Backward Effects*.—The left ventricle becomes hypertrophied in consequence of the increased effort required to force the blood through the stenosed orifice; but so long as the mitral valve remains competent—and it usually does so even in advanced cases—there are no prominent lung symptoms, or other signs of venous engorgement.

B. *Forward Effects*.—The pulse is small but of good tension, slow and regular. When the stenosis is considerable, symptoms due to defective blood-supply to the brain are sometimes observed.

AORTIC REGURGITATION.

A. *Backward Effects*.—The left ventricle becomes hypertrophied and dilated, and in consequence of the dilatation of the cavity and the impaired nutrition of the cardiac muscle, mitral regurgitation is common towards the later stages of the case. So long as the mitral valve remains sound there are no prominent lung symptoms or other signs of venous engorgement; should the mitral give way the symptoms characteristic of mitral regurgitation are super-added to the symptoms which I must now describe.

B. *Forward Effects*.—At each systole of the dilated and

Description of Fig. 58.

Diagrammatic representation of the effects of a mitral lesion upon the venous circulation. The numbers and letters are the same as in fig 3. (See description page 5.) The arrows indicate the direction of the backward current.

hypertrophied left ventricle, a large quantity of blood is propelled into the arterial system, which is therefore rapidly and fully distended; but, in consequence of the leak at the aortic orifice, this distention of the arterial system is not maintained. The pulse is highly characteristic, presenting the jerking, visible, collapsing, water-hammer character which was so ably described by the late Sir Dominic Corrigan (Corrigan's pulse.) It is generally quicker than in health. The aortic and dicrotic wave is, as a rule, feebly marked or absent, and the sphygmographic tracing is, in some cases, characteristic. When the regurgitation is free, the artery may be very empty during the ventricular diastole. The face is generally pale, and usually presents an anxious expression. In advanced cases, attacks of syncope are common, and the general condition of nutrition may be considerably impaired. Pain of an angina-like character is frequently observed. The amount of alteration in the pulse, more especially the degree of distention during the ventricular diastole; the extent of the dilatation of the left ventricle; and the condition of the mitral valve—whether competent or not—are points of great importance in estimating the gravity of this lesion.

Figure 59 represents in a diagrammatic manner the effects of aortic lesions upon the circulation.

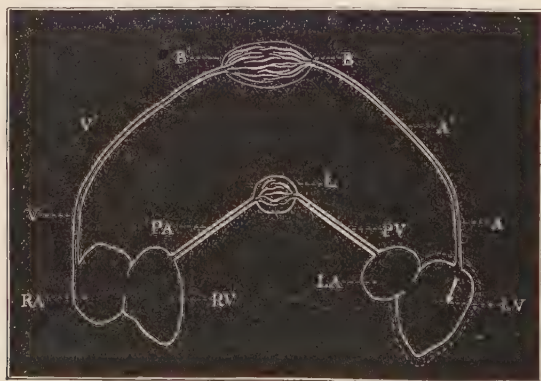


FIG. 59.—Representation of the effects of a lesion of the aortic valves (aortic regurgitation in this case) on the heart and circulation. The letters have the same significance as in fig. 57. The left ventricle is hypertrophied and dilated; the mitral valve is as yet competent.

PULMONARY AND TRICUSPID LESIONS.

The effects produced by pulmonary and tricuspid lesions will be readily understood after what has been already stated in describing the effects of mitral lesions.

Pulmonary lesions—which are extremely rare, except as congenital conditions—produce alterations in the right cavities of the heart and engorgement of the systemic venous circulation.

Tricuspid regurgitation (which is frequent in the advanced stages of mitral lesions, and which may also arise, as I have previously pointed out, from primary disease of the lungs, such as emphysema and cirrhosis) is always attended with marked signs of systemic venous engorgement. Venous pulsation in the neck, synchronous with the contraction of the right ventricle, is very characteristic of this condition; and in some cases true pulsation in the liver, the result of a back-wash through the inferior cava, is observed.

2. *The amount of compensation which can be produced.*—In all valvular lesions there is an attempt on the part of nature to meet the difficulty. The extent to which the lesion can be compensated, and the probable time during which this compensation can be maintained, are most important points in estimating the gravity of the lesion. The capability of compensation depends upon:—

(a) *The general reparative powers of the individual.*—A valvular lesion of moderate extent, occurring in an individual whose tissues are prone to degenerate, or already in a state of decay, is of graver significance than a much more extensive lesion in a person whose tissues are otherwise healthy.

(b) *The special reparative power of the cardiac muscle.*—So long as the cardiac muscle remains sound, and the hypertrophy is good, compensation is well maintained, and the symptoms are at a minimum. But whenever the muscular nutrition fails, or dilatation occurs, the compensation gradually fails, and serious symptoms arise. The structural soundness therefore of the cardiac muscle, and the presence or absence of dilatation are points of great importance in estimating the gravity of the lesion.

(c) *The age of the patient.*—Other things being equal, the younger the patient, the better the prognosis, for in young persons the reparative powers, and therefore the capabilities of compensation, are greater than in old people.

(d) *The habits and surroundings of the patient.*—Valvular lesions are (other things, such as the condition of the tissues, being equal) much less serious in persons in comfortable circumstances than in those who are obliged to struggle for existence, and lead laborious lives. Indeed, as we shall see when I come to speak of the treatment of these affections, rest—so far as is possible—to the damaged organ is the first and most important point to be attended to. The amount of work required of the damaged heart, is a very important element in the prognosis.

(e) *The mental temperament of the patient* is of considerable importance. Valvular lesions are less serious in persons of a quiet and placid disposition than in persons of an excitable and anxious temperament.

(f) *The ætiology of the lesion.*—Valvular lesions of rheumatic origin are, as a rule, less serious than those which result from other causes. This is probably owing to the facts, that non-rheumatic valvular lesions are often due to degenerative processes, and that persons thus affected are, as a rule, older than those affected with lesions which can be directly traced to acute rheumatism.

3. *The form of valvular lesion.*—We know, as the result of clinical experience, that some valvular lesions are more serious than others. Dr Walshe gives the following as the order of relative gravity, estimating the gravity not only by the *ultimate* lethal tendency of the different lesions, but also by the amount of complicated miseries which they inflict. (This order can only be looked upon as approximate. I shall afterwards have to point out many modifications in it. There are, for instance, many different causes of mitral regurgitation, some of which are eminently curable; others almost certainly fatal. Dr Walshe evidently alludes to the more serious forms. So again, tricuspid regurgitation may be a temporary and curable condition, though in many

cases it is the most unfavourable of all cardiac valvular lesions):—

1. Tricuspid regurgitation (most grave).
2. Mitral regurgitation.
3. Mitral constriction.
4. Aortic regurgitation.
5. Pulmonary constriction.
6. Aortic constriction (least grave).

The relative gravity as regards their tendency to produce sudden and instantaneous death is, however, quite different. Aortic regurgitation stands prominently out as *the* valvular lesion which often gives rise to immediate death, the fatal result being due to syncope; while the other valvular lesions have no direct tendency to produce immediate death.

4. *Whether the lesion is progressive or stationary.*—This is an extremely important point, and it is to be determined—

(a) By close observation of the case, and noting the condition of the patient from time to time.

(b) By comparing the duration of the case and the effects which the lesion has already produced on the heart and circulation.

(c) By taking into account the nature of the morbid process. We know as the result of clinical and pathological observation, that valvular lesions, due to degenerative processes, are less likely to remain stationary than those which result from simple inflammation (endocarditis).

(d) By reference to the valve which is affected, and the manner in which it is affected. Mitral regurgitation, for example, is in many cases curable, but aortic regurgitation is not.

5. *The associated pathological conditions.*—This is a point of the very greatest importance. In all cases in which the tissues are prone to degenerate, the prognosis is bad; in fat, flabby individuals, a lesion—other things being equal—usually advances with more rapidity, and proves more speedily fatal than in spare and thin people. The presence of kidney disease, or of any other organic lesion, adds, of course, very materially to the gravity of the case.

The points which enable us to determine the extent and

gravity of the lesion will be considered in further detail under the prognosis of the individual valvular lesions.

THE PHYSICAL EXAMINATION OF THE AORTA AND THE
GREAT BLOOD VESSELS.

After having ascertained the condition of the heart itself, we must next determine the physical condition of the aorta and great blood-vessels. In actual practice it is customary and convenient to examine the condition of the arch of the aorta, and of the heart, at one and the same time ; when inspecting the præcordia, for example, to inspect at the same time the parts of the chest which lie superficial to the aortic arch ; and so on with palpation, percussion, and auscultation. In ordinary cases it is not customary to make a detailed examination of the great branches of the aortic arch, or of the descending thoracic or abdominal portions of the aorta. When, however, there is any reason to suspect disease of these structures, their condition must be carefully and methodically examined.

In order to ascertain the physical condition of the aorta (both its thoracic and abdominal portions) and of its branches, we employ the same means of investigation which I have already described in speaking of the physical examination of the heart :—

Firstly we inspect, palpate, percuss, and auscultate the parts superficial to the vessel.

Secondly, we investigate the condition of the circulation and of the circulatory organs in front and behind ; when we are investigating the condition of the aortic arch, for instance, we direct special attention on the one hand to the condition of the pulse in the different branches arising from it (*i.e.* we observe the comparative condition of the pulse in the two radials and carotid arteries), and on the other to the state of the heart and venous circulation.

Thirdly, we pay special attention to the physical condition of the organs and parts adjacent to the vessel. This is a point of great importance, for the chief pathological conditions in the aorta and its branches, which we are able to ascertain

by means of physical examination, are dilatations (simple and aneurismal) and the chief symptoms and physical signs in these conditions are often due to the pressure of the dilated blood vessel upon adjacent parts, and to the displacements which are caused thereby.

INSPECTION APPLIED TO THE EXAMINATION OF THE
THORACIC AORTA.

The patient should be placed in a good light, and the surface of the chest carefully inspected, more especially those parts which are superficial to the course of the aorta, and at which the vessel comes nearest to the surface (the sternal end of the second right interspace in particular). But in order that this and other points connected with the physical examination of the thoracic aorta, and the symptomatology of its diseases, may be thoroughly understood, it will perhaps be well for me to describe briefly the anatomical course of the vessel and its relations, which are of importance from a practical and clinical point of view.

Anatomical course and relations of the thoracic aorta.

The thoracic aorta arises at the junction of the third left costal cartilage with the sternum, *i.e.* nearly opposite the point of union of the upper and lower sternal regions, and terminates in the abdominal aorta beneath the pillars of the diaphragm, or more exactly, on the anterior surface of the last dorsal vertebra. The thoracic aorta has been divided, for descriptive purposes, into two portions—the aortic arch and the descending thoracic aorta.

The *aortic arch* is described as consisting of three parts—the *ascending*, *transverse*, and *descending* portions.

The *ascending portion* of the aortic arch arises from the base of the left ventricle, on a level with the lower border of the third left costal cartilage, at its junction with the sternum, and passes upwards and to the right until it reaches the upper surface of the second right costal cartilage at its junction with the sternum (occasionally encroaching upon the inner edge of the first interspace), where it terminates in the transverse

portion. For the greater part of its course it is enclosed in the membranous sac of the pericardium. In the first part of its course it is deeply situated, being covered by the root of the pulmonary artery, and being closely related posteriorly to the cavity of the left auricle. After emerging from under cover of the pulmonary artery, it comes in close relationship with the sternum and second right costal cartilage, being separated from these structures by the sac of the pericardium, the cellular tissue and fat of the mediastinum, and (when the lungs are expanded) by the thin anterior margin of the lungs, more especially of the right lung. The superior vena cava lies in contact with it on the right side; the pulmonary artery diverges from it on the left side, while behind it is placed the root of the right lung. The cardiac plexus and many of its branches, as they proceed to their terminations in the coronary plexuses, are closely related to the root of the aorta. This is a connection which is of great practical importance; while the facts that the coronary arteries arise from the root of the aorta, and that the root of the aorta is directly continuous with the aortic valves, and therefore with the heart, are points the importance of which, from a practical clinical point of view, is self-evident.

The transverse portion of the arch of the aorta commences at the junction of the upper edge of the second right costal cartilage with the sternum, and crosses almost horizontally through the upper sternal region, on the level of the first interspace, passing backwards and downwards deeply into the chest. The transverse portion terminates in the descending portion of the aortic arch, at the lower border of the left side of the body of the fourth dorsal vertebra.

The transverse portion of the aortic arch is separated from the surface of the front of the chest by the mediastinal fat and connective tissue, by the remains of the thymus gland, during inspiration by a small portion of the right pleura and anterior border of the right lung, and by the left pleura and anterior border of the left lung. The left pneumo-gastric, the left phrenic and superficial cardiac nerves, and the left superior

intercostal vein, cross in front of this portion of the vessel. On the right side, at its origin, it is closely related to the superior vena cava, the right pneumo-gastric and phrenic nerves. From its upper convex surface arise the great blood vessels destined for the head and neck and upper extremities (the innominate, left common carotid, and left subclavian arteries), and in close relation with its upper surface lies the left innominate vein. In the concavity beneath its lower surface the bifurcation of the pulmonary artery and the cardiac plexus of nerves are situated; the recurrent branch of the left pneumo-gastric winds round the concave lower surface of the vessel to which the obliterated ductus arteriosus is attached. Posteriorly the transverse portion of the arch of the aorta is in close contact with the left recurrent laryngeal nerve, as it ascends through the thorax to the neck, with the trachea and bifurcation of the bronchi, the œsophagus, thoracic duct, and more deeply with the bodies of the vertebræ.

The *descending portion* of the aortic arch commences at the junction of the bodies of the fourth and fifth dorsal vertebræ, and passes downwards in close contact with the left side of the body of the fifth dorsal vertebra, until it terminates at the lower end of the body of that vertebra, in the descending thoracic aorta.

The *descending thoracic aorta* passes downwards in contact with the spinal column; in the upper part of its course it lies on the left side, in the lower part of its course on the anterior surface of the bodies of the vertebræ. It terminates in the abdominal aorta at the level of the twelfth dorsal vertebra. On the right side of the vessel lie the thoracic duct and the large azygos vein. On the left side the vessel is covered by the left pleura and left lung. In front of it are placed the root of the left lung and the posterior surface of the pericardium, and, therefore, the posterior surfaces of the left auricle and left ventricle. The œsophagus is at first on the left side of the aorta, lower down it makes its way in front of the vessel. At its termination the thoracic aorta is enclosed in the opening formed by the crura of the diaphragm.

In health, inspection gives no information as to the condition of the thoracic aorta; in other words, neither pulsation nor prominence can be seen on those parts of the surface of the chest which correspond to the position of the vessel underneath. When, however, the vessel is diseased, more especially when it is affected with aneurismal dilatations, local prominence and pulsation at the part of the chest, corresponding to the position of the aneurismal dilatation, are often observed. In order to detect slight elevations of the chest wall and slight pulsations, a special method of inspection is necessary; instead of placing himself in front of the patient and looking straight on to the surface of the chest, the observer should place himself at one side of the patient—the opposite side to that from which the rays of light are proceeding—and should then bring his eye to the same horizontal plane as the surface of the chest which he wishes to examine. If the illumination is good, slight elevations and pulsations, which might easily escape observation by the ordinary or full-face method of inspection, can by this means be readily detected.

The most frequent position for aneurismal or aortic pulsation is the second right interspace close to the sternum; for, in the first place, the ascending portion of the aortic arch is more liable to be affected by aneurismal dilatation than any other part of the vessel; and, in the second, comparatively small aneurisms are apt to produce bulgings at this spot, for the aorta is here very superficial. Aneurisms may, however, affect the vessel in any part of its course, and may ‘point’ at any part of the chest with which the dilated vessel comes in contact. Further details on these points will be afterwards given. (See Chapter VIII.)

In some cases of aortic dilatation and aneurism, pulsation is seen in the supra-sternal notch.

PALPATION APPLIED TO THE EXAMINATION OF THE THORACIC AORTA.

In health the pulsation of the aorta can sometimes be felt in the supra-sternal notch, but in no other position.

When the vessel is dilated, more especially when a saccular aneurism is in contact with the chest wall, pulsation can often be felt over the position of the sac. When the chest wall is bulged forwards, the alteration in level can often, of course, be detected by the finger as well as by the eye.

To detect slight superficial pulsations (those pulsations, for example, which are produced by an aneurismal sac which is in contact with the chest wall, but which has not as yet produced perforation or prominence), the fingers of the right hand should be lightly placed over the seat of the suspected dilatation. In some cases in which the aneurism is deeply seated, and in which there is no superficial pulsation, forcible, deep-seated and expansile pulsation can sometimes be detected by forcibly compressing the chest during expiration, between the two palms, one hand being placed on the front and the other on the back of the chest.

When the transverse portion of the arch is dilated or aneurismal, pulsation can often be very readily felt in the supra-sternal notch,—the head should be bent well forwards so as to relax the sterno-mastoids, and the forefinger of the right hand placed in the supra-sternal notch, and pushed downwards behind the manubrium sterni.

Vibratile thrills can be felt over the course of the aorta, more especially over the position of the ascending portion of the aortic arch, in some cases of dilatation and aneurism.

PERCUSSION APPLIED TO THE EXAMINATION OF THE THORACIC AORTA.

Percussion of the healthy aorta yields only negative results. At its origin the vessel is overlapped by the pulmonary artery, and even when the percussion note is impaired over the part of the chest corresponding to the root of the aorta, as it is, for example, in very full expiration, the dulness is of course derived from both vessels. After the aorta emerges from under cover of the pulmonary artery, although it lies close under the sternum, its position and outline in health cannot be definitely determined by means of percussion, for on percussion over the manubrium sterni in the healthy

condition, a more or less resonant note is obtained. In the subsequent part of its course the vessel is so deeply situated as to preclude the possibility of detecting its presence by this means of investigation.

When the aorta is dilated or affected with aneurism, percussion often yields most important results; the extent and position of the dulness depend, as we shall afterwards see, upon the size and position of the sac, more especially upon its relation to the lung and to the chest-wall. The percussion resistance is often increased over a dilated or aneurismal aorta, the resistance being greatest in those cases in which an aneurismal sac, filled with laminated clot, lies in close contact with the chest wall.

AUSCULTATION APPLIED TO THE EXAMINATION OF THE THORACIC AORTA.

In health two sounds can usually be heard when the stethoscope is placed over the course of the thoracic aorta. Over the ascending portion of the aortic arch these sounds closely resemble the normal heart sounds, only that they are less loud, the first sound more especially being weaker than the first sound as heard over the heart itself. Over the descending portion of the thoracic aorta the sounds are usually very faint and distant.

The sounds heard over the aorta undergo the same quantitative and qualitative alterations, which have previously been described in detail in treating of the modifications of the cardiac sounds; and since the sounds, which are heard over the aorta, more especially those heard over the ascending and transverse portions of the aortic arch, are for the most part composed of the sounds produced within the heart, propagated through the aorta to the ear of the observer, it follows, that alterations of the heart sounds (more particularly those alterations which are due to disease of the aortic valves), will be heard over the course of the aorta. In other words, it is essential to remember that murmurs heard over the aorta are very frequently due to disease of the cardiac valves, and not to disease of the aorta.

Alterations of the sounds heard over the aorta (both quantitative and qualitative modifications) may, however, be due to disease of the aorta itself; and in cases of this description, the heart sounds, as heard over the heart itself, may be perfectly normal. When, for example, an aneurism of the aorta approaches the surface of the chest, the aortic or cardiac sounds are much more clearly and distinctly heard over that part of the chest, which corresponds to the position of the aneurism, than they would be under normal circumstances. In some cases, the change is merely a quantitative one; in others, a murmur is heard. But I must defer the more detailed description of the character of the sounds in cases of this nature until I come to treat of aneurismal and other dilatations.

In other cases in which the aorta itself is healthy, the aortic sounds appear to be louder than in health, in consequence of the fact that they are more easily conducted to the ear than in the normal condition. When, for example, a solid tumour lies in contact with the aorta on the one hand and with the chest wall on the other, an *apparent increase* of this description is frequently observed. On the other hand, *apparent diminution* is produced by all those conditions, such as emphysema, for example, which interfere with conduction. I need not, however, go into details with regard to these points, but must refer the reader to what has been already stated with regard to the modifications of the heart sounds.

When the aorta is compressed and constricted, a systolic murmur may be generated.

THE EXAMINATION OF THE PERIPHERAL ARTERIES.

In all cases of cardiac and arterial disease it is of the greatest importance to observe the condition of the peripheral and superficial arteries, and to ascertain the manner in which the circulation is being carried on in these vessels.

By observing the colour of the skin we obtain important information as to the condition of the peripheral circulation (both arterial and venous), but this point has been already considered in treating of the physiognomy of cardiac cases.

The radial is the artery which is usually examined, but the condition of the carotids, temporals, brachials, etc., and the character of the pulse in these vessels should also be noted.

I must, therefore, now describe the manner in which we observe the pulse and the characters which it presents both in health and disease. And in order that the subject may be thoroughly understood (for it must be remembered that important modifications of the pulse are met with independently of any cardiac or arterial disease) I shall take a somewhat comprehensive view of the subject, and shall not limit the description altogether to the alterations which are met with in disease of the heart and aorta.

THE EXAMINATION OF THE PULSE, INCLUDING A DESCRIPTION OF THE SPHYGMOGRAPH.

At each contraction of the left ventricle, some five or six ounces of blood are suddenly propelled into the aorta, and a blood-wave is generated, which is rapidly propagated through the arterial system. The temporary distention of the arterial walls which is caused by this blood-wave, can be felt by the finger or measured by the sphygmograph, and is termed the pulse.

The exact character of the arterial expansion, *i.e.* of the pulse, varies in different cases, and depends partly upon the mode of contraction of the left ventricle, and the amount of blood which it propels into the aorta, and partly upon the condition of the arterial system. It is evident therefore that in the examination of the pulse we have an important means of investigating *the condition of the heart* and of *the arterial system*; and since the condition of the arterial system is to a large extent regulated by the vaso-motor nerve apparatus, we are enabled, by the observation of the pulse, to obtain in many cases valuable information as to the *condition of the nerve tone* (*i.e.* the general tone of the system); the frequency and strength of the pulse being chiefly valuable in this respect.

¹ *Text-Book of Physiology*, by Professor M. Foster, p. 157.

The radial is the artery which is usually examined, and in speaking of *the pulse* the radial pulse is meant; but, in cases of cardiac and arterial disease, the condition of other vessels (the carotids, temporals, brachials, etc.) should be noted.

MODE OF OBSERVING THE PULSE.

We observe the characters of the radial pulse by means of—

- (1) the finger (*palpation*);
- (2) the eye (*inspection*);
- (3) the sphygmograph.

(1) PALPATION, OR THE EXAMINATION OF THE PULSE
BY THE FINGER.

The correct observation of the exact characters of the pulse by the finger is a matter of extreme difficulty, and is only acquired by long practice. Since, however, it is *the* method which is not only always available, but which yields far more useful and important information than any other, the student should spare no pains to make himself master of it. Two or three fingers should be applied over the artery where it becomes superficial at the lower end of the radius, and the condition of the pulse noted as regards—

- (a) its frequency;
- (b) its rhythm;
- (c) its volume;
- (d) its compressibility or tension;
- (e) the special characters of each pulse wave (celerity, dicrotism, etc.); the condition of the vessel (in respect to its fulness) during the diastole of the ventricle, *i.e.* between the beats;
- (f) the condition of the arterial coats.
- (g) In cases of suspected aneurism or intra-thoracic tumour a comparison of the two radial pulses should be made.

(2) INSPECTION OF THE PULSE.

In well-nourished individuals the radial pulse is hardly, if at all, visible when the circulation is tranquil; but in emaciated subjects, and during cardiac excitement, its pulsation can often be distinctly seen. Pulsation is very visible in certain cases of disease, notably in conditions of high tension; in atheroma, where the artery stands out as a rigid, tortuous cord; and in aortic regurgitation, where the pulsation is visible, jerking, and collapsing, and the artery tortuous (the locomotive pulse).

In conditions of vaso-motor relaxation with excited action of the heart, the pulse in the peripheral vessels (the radial for example) may present the visible, jerking, collapsing character of aortic regurgitation; but the marked (visible, jerking, collapsing) pulsation in the large vessels, *e.g.* the carotids, which is so characteristic of aortic regurgitation, is not observed.

(3) THE EXAMINATION OF THE PULSE BY MEANS
OF THE SPHYGMOGRAPH.

The exact characters of the pulse, *i.e.* of its individual waves, and of their relationship to one another, are graphically demonstrated by means of the sphygmograph. The instrument is chiefly useful as an indicator of the manner in which the circulation is being carried on, and of the general condition of the vascular system; it sometimes gives important diagnostic evidence, as in the earlier stages of chronic Bright's disease (especially the cirrhotic kidney), and in some aneurisms; but it is comparatively useless and superfluous as a means of diagnosing individual cardiac affections. But although its (direct) diagnostic value is limited, it sometimes enables us to form a more correct opinion than we could otherwise of the severity of a lesion or case; in pneumonia, and typhoid fever for example, it may afford most useful prognostic information, and the same may be said of many cardiac affections. I must repeat, however, that the examina-

tion of the pulse by means of the sphygmograph is altogether secondary and subordinate to the ordinary examination by the finger.

FORMS OF SPHYGMOGRAPH.

Mahomed's modification of Marey's¹ Sphygmograph, and Dudgeon's² Sphygmograph, are the best forms. (Dr W. J. Fleming of Glasgow introduced a 'simple form of transmission sphygmograph' some years ago. I have not had an opportunity of using this instrument, which is described and figured in the *Journal of Anatomy and Physiology*, vol. xii. p. 144).

For home practice or hospital work I prefer Mahomed's modification of Marey's instrument, as I think it permits of more accurate adjustment of the pressure; but for general practice Dudgeon's instrument is undoubtedly most convenient; it is extremely portable, easily applied in any position of the patient, and is only one-third of the cost of the larger instrument. With it excellent tracings may be obtained, and its inventor claims that it gives a more accurate and natural representation of the up-stroke than can be obtained by Marey's instrument.³

¹ This instrument is made by Krohne and Sesemann, 8 Duke Street, Manchester Square, London.

² Dr Dudgeon's instrument is made by Mr J. Ganter, and may be obtained through any instrument-maker.

³ Dr Dudgeon claims the following advantages for his instrument :—

1. It magnifies the movements of the artery in a uniform degree, viz. 50 times.
2. The pressure of the spring can be regulated from 1 to 5 ounces.
3. It requires no wrist-rest, and may be used with equal facility whether the patient is standing, sitting, or lying.
4. With it a tracing of the pulse can be made almost as quickly as the pulse can be felt with the finger.
5. Its sensitiveness is so great that it records the slightest deviation in form or character of every beat.
6. Its construction is so simple, that if accidentally broken any watchmaker can repair it.
7. It is so small ($2\frac{1}{2}$ by 2 inches), and it is so light (4 oz.), that it can easily be carried in the pocket.
8. It is only one-third of the price of the imperfect and cumbrous instruments hitherto offered to the profession.

DESCRIPTION OF THE SPHYGMOGRAPH.

Mahomed's modification of Marey's Sphygmograph consists of—

1. A steel spring, A (see figs. 60, 61, and 62), which rests on the artery, and which moves up and down with each movement of the vessel.

One end of the spring, A' (see figs. 61 and 62), is so attached by a hinge to the framework of the instrument that vertical (up and down) movement is alone permitted. To the under surface of the free end of the spring an ivory pad, A'', is fixed. The ivory pad rests on the artery.

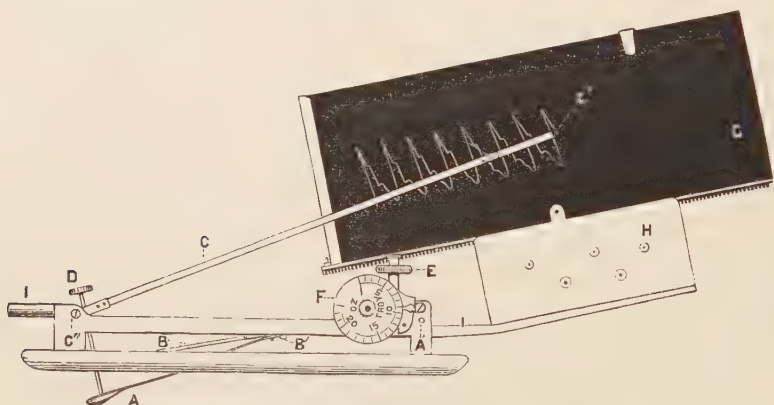


FIG. 60.—*Mahomed's Modification of Marey's Sphygmograph.*

A, points to the steel spring; A', to its point of attachment to the framework; B, the first lever; B', its point of attachment to the steel spring A; C, the writing lever; C', its free end, which carries a pen; C'', its point of attachment to the framework; D, the screw for bringing the turned up free end of lever B in contact with lever C; E, the screw for increasing the pressure; F, the dial on which the amount of pressure is indicated; G, the slide carrying the strip of smoked paper; H, the clockwork, which is wound up by a screw on the opposite side to that shown in the figure; I, I, parts of the instrument over which the straps, which fix it to the arm, are passed.

2. An arrangement of levers, by means of which the movements of the steel spring (*i.e.* of the artery) are magnified and recorded on a piece of smoked paper.

There are two levers. The lower one, B, is so hinged by its proximal end, B', to the middle of the steel spring, that up and down movement is alone permitted. The free end of this lever (B'', figs. 61 and 62) is

turned up at a right angle, and ends in a rounded or knife-shaped edge. Through the free extremity of this lever, B (just before it terminates in the turned up end, B''), a screw D is passed. The point of the screw is always (by the force of gravity) in contact with the steel spring, which rests on the artery. And since the screw D and the lever B are practically the same, it follows that every movement of the artery is of necessity communicated to the turned up extremity B'', of the lever B. The object of the screw D is to raise or lower the turned up edge of the lever B, so that it may, whatever the position of the steel spring, be in proper contact with the writing lever C, in other words, in order that it may always communicate the movements of the steel spring, *i.e.* of the artery, to the writing lever C.

The second lever, C, is so fixed at its distal end, C'', to the framework of the instrument, that vertical movement is alone possible. The free end of this lever, C', carries a pen which records its movements on a strip of smoked paper propelled past it at a fixed rate by means of clockwork. In order that the movements of the steel spring may be communicated to the writing lever, the knife-edge of lever B must be in contact with lever C, as shown in figure 61. This is effected, as has been already stated, by alteration of the screw D.

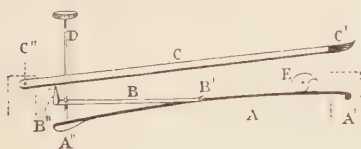


FIG. 61.

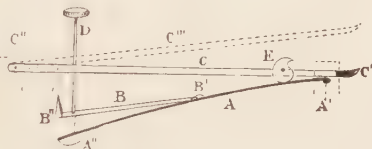


FIG. 62.

FIG. 61.—Scheme showing the essential parts of the instrument *when in working order*—*i.e.* the turned up knife-edge B'' of the short lever in contact with the writing lever C. Every movement of the steel spring, at A'', *i.e.* of the artery, will, when the knife-edge, B'', is in this position, be communicated to the writing lever. The letters have the same significance as in fig. 60.

N.B.—(The framework of the instrument has been removed).

FIG. 62.—Scheme showing the essential parts of the instrument *after increase of the pressure*. The knife-edged B'' is no longer in contact with the writing lever, and the movements of the steel spring A'', *i.e.* of the artery, are no longer communicated to it. In order to put the instrument into working order the knife-edge B'' must be raised to the position indicated by the dotted lines. This is effected by means of the screw D.

3. An arrangement by which the amount of pressure exerted by the steel spring on the artery can be regulated and measured. This is a most important part of the instrument, and is the modification made by Mahomed. It consists of an eccentric, E, by depression of which, as shown in fig. 62, a definite degree of pressure can be made upon

the steel spring. The amount of pressure exerted is shown on a dial (F, in fig. 60) in ounces troy. The eccentric (see figs. 60, 61, 62) is depressed by turning the screw E (see fig. 60).

4. A clockwork, H, which propels at a fixed rate a slide G, to which a strip of smoked paper is attached.

5. A framework to which the various parts of the instrument are fixed, and by means of which the instrument is fastened to the arm by straps (K, K, fig. 65).

Dudgeon's Pocket Sphygmograph (see fig. 63) consists of:—

1. A steel spring, A (see fig. 64), which rests upon the artery, and moves up and down with each movement of the vessel.

One end of the spring (a) is firmly attached to the framework of the instrument, the other (C) is turned up at a right angle. To the under extremity of the steel spring a button, B, which rests on the artery, is fixed; and to the turned up extremity C a short rod (D) is firmly attached.

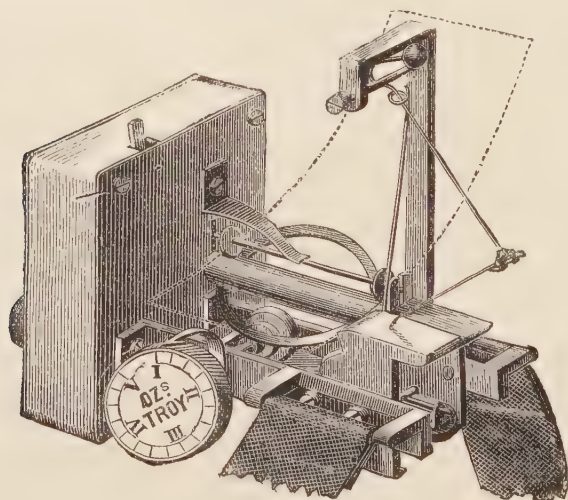


FIG. 63.—Dudgeon's Sphygmograph.

2. An arrangement of levers by means of which the movements of the steel spring, *i.e.* of the artery, are

magnified and recorded on a strip of smoked paper propelled by clockwork.

At right angles to D, and connected with it by the axle E, rises the upright stem F. Every upward movement of the steel spring causes the upright F to move forwards. At the top of F is a loop in which a rod K lies. This rod is connected at the axle H with a bent rod having a counterpoise I. When the upright F makes a forward movement, the oblique rod K also swings forwards by the weight of its counterpoise.

To the lower end of K the needle L is attached by the hinge M, and its point describes on the smoked paper, which is propelled by the clockwork machinery at a uniform velocity, a graphic representation of the movements communicated to it.

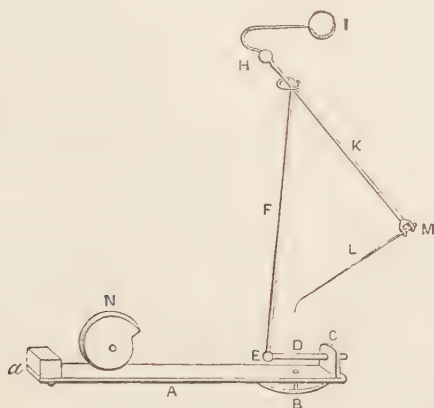


FIG. 64.—Scheme showing the different parts of Dudgeon's Sphygmograph.
The description of the figure is given in the text.

3. An eccentric, by means of which the pressure can be increased from one to five ounces.

4. A clockwork, by means of which a strip of smoked paper is propelled at a fixed rate under the writing lever.

5. A framework, by means of which the instrument can be attached to the arm.¹

¹ For further particulars respecting Dr Dudgeon's instrument, see his book, *The Sphygmograph.*

DIRECTIONS FOR TAKING A SPHYGMOGRAPHIC TRACING
WITH MAREY'S INSTRUMENT.¹

1. *Place the Patient in proper position.*—He should be seated by the side of a low table, his arm resting on the pad (a double inclined plane), as represented in fig. 65, the fingers semiflexed into the palm.

If the fingers are quite extended the artery is too much stretched, and jerking movements of the tendons, which interfere with the tracing, are apt to occur. If the fingers are quite flexed, the rigidity of the tendons prevents the perfect application of the instrument.

The position should be as easy as possible, for it is essential that the arm be kept at perfect rest. The shirt sleeve should be turned up; and it must be loose, lest it interfere with the circulation through the arm.

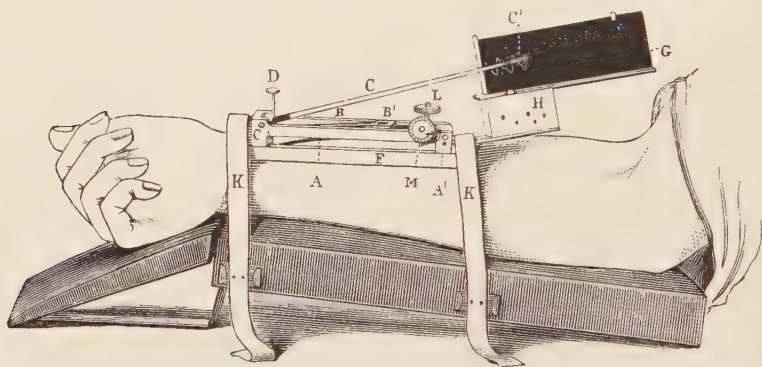


FIG. 65.—Marey's Sphygmograph applied to the wrist.

2. *Mark the exact position of the artery with ink or pencil.*—The ink line should be prolonged to the ball of the thumb, *i.e.* below the instrument when *in situ*. By this means we can, without removing the sphygmograph, ascertain if the ivory pad is still in proper position, *i.e.* exactly over the artery.

¹ For further information on the subject the student is referred to Dr Mahomed's papers in the *Medical Times and Gazette* (January 20th, 1872, and following numbers), and in *Gant's Surgery* (vol i., page 52), to which I am indebted for much of my information on the subject, and from which some of the following rules for the application of the instrument are derived.

Before applying the sphygmograph, the harmless nature of the procedure must be explained to the patient, for it is of the greatest importance to avoid anything which will excite or disturb the action of the heart. Some persons become considerably agitated, thinking that an operation is about to be performed. (One of my patients left the hospital rather than have the instrument applied.) In such cases a preliminary application to a fellow-patient or nurse is advisable.

3. *Apply the instrument*, having previously screwed up the clockwork and placed the pressure at zero.—The ivory pad must be *accurately* applied over the *very centre* of that part of the artery which lies at the inner side of the styloid process of the radius. By compressing the vessel at this spot, where it is superficial and rests upon bone, we can be quite certain that the entire pressure of the spring will be exerted upon it. The instrument is then firmly strapped to the arm. The straps should be unyielding—not elastic. The slide carrying the strip of smoked paper is next to be fitted into the frame. Care must be taken that the paper is firmly and evenly stretched. This is best effected by first accurately fitting it and doubling its edges over the frame, then removing and smoking it over a piece of burning camphor, and finally fitting it to the frame again. The paper should not be over-smoked, and the point of the pen must not press too heavily against it, or friction will prevent free movement, and the tracing will be imperfect.

4. *Adjust the pen*.—The point of the pen is then (by means of the screw D, see figs. 60 and 62) to be brought level with the centre of the strip of smoked paper, as shown in figs. 60 and 65.

5. *Regulate the pressure* by means of the screw L, until the maximum amount of movement of the writing lever is obtained.¹ There is a certain pressure, depending upon the amount of expansion which is going on in the artery, at which the tracing is best marked. If the pressure is too

¹ The reader must not forget that every alteration of the pressure, *i.e.* every alteration in the position of the steel spring necessitates a fresh adjustment of the writing lever by means of the screw D.

little, *i.e.*, if the steel spring just touches the artery in its expanded state, the rise of the lever will be insignificant ; if, on the other hand, the pressure be too great, the artery cannot expand to its full amount, and the tracing will be imperfect,—the up stroke shortened (see fig. 66), and the perfect development of the secondary waves of the tracing interfered with.

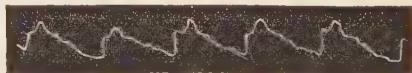


FIG. 66.—*Tracing taken under too great pressure.*

The up-stroke is cut short, and the perfect development of the tracing prevented.

If the tracing is satisfactory, the name of the patient, the date, the nature of the disease, and the amount of pressure which is required for (*a*) the perfect development of the tracing, and (*b*) the complete obliteration of the pulse in the artery (the latter being the gauge of the strength of the pulse) should be inscribed upon the slip of smoked paper by means of a needle or other fine-pointed instrument, and the tracing rendered permanent by dipping it in a rapidly drying varnish.¹

Character of a good tracing.—In a good tracing the apex (*b*, fig. 67) is pointed ; and the best tracing is that in which the

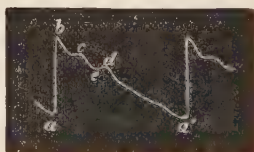


FIG. 67.—*Normal Pulse Tracing.*

up-stroke is tallest and the apex most pointed. It occasionally happens that the apex is rounded, as in some aneurisms

¹ The varnish recommended by Mahomed is made by macerating an ounce of gum benzoin in five ounces of rectified spirit ; the mixture, which should be frequently agitated, is allowed to stand for two days, and the clear liquor then poured off from the insoluble constituents of the gum. Dr Dudgeon uses the crystal varnish of photographers, or a varnish consisting of one ounce of gum damar and six ounces of rectified benzoline

(see figs. 68 and 69), and in a few cases of aortic stenosis (see fig. 70); but this is so extremely rare, that a tracing in which the apices are rounded should always be regarded as imperfect, unless it has been verified by repeated and careful readjustments of the instrument, and by repeated alterations of the pressure.¹



Pressure $2\frac{1}{4}$ oz.

FIG. 68.—*Aneurism of Left Axillary Artery* (left radial tracing).—L. G., æt. 63, admitted to the Newcastle Infirmary 7th March 1878, with a large aneurism of the left axillary artery. The apex is rounded; all the curves are obliterated.



Pressure 3 oz.

FIG. 69.—*Aneurism of Left Subclavian* (left radial tracing).—J. M., æt. 50, admitted to Newcastle Infirmary 5th September 1878: all the waves in the left tracing are obliterated.



Pressure $1\frac{1}{2}$ oz.

FIG. 70.—*Aortic Stenosis*.—J. B., æt. 51, admitted to Newcastle Infirmary 29th November 1878, suffering from anæmia and dropsy. There was a well-marked aortic systolic murmur; the left ventricle was not hypertrophied. The pulse tracing seems to show that the murmur was organic.

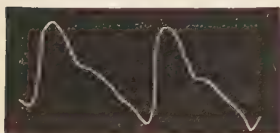


FIG. 71.—Pressure $1\frac{1}{2}$ oz.

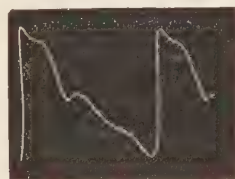


FIG. 72.—Pressure $2\frac{3}{4}$ oz.

FIG. 71.—*Aortic Stenosis and Dilated Aorta*.—J. C., puddler, æt. 25, admitted to Newcastle Infirmary 20th February 1879. The patient had been under observation for four years previously. Marked thrill and loud systolic murmur over base of heart and over aortic region. Heart moderately hypertrophied. Pressure = $1\frac{1}{2}$ oz.

FIG. 72.—Tracing taken from the same patient with a slightly increased pressure; the apex is now pointed.

¹ In most cases of aortic stenosis a pointed apex can be obtained by careful adjustment and regulation of the pressure. (See figs. 71 and 72.)

Dr Galabin points out that 'if the tracing of any pulse taken at a low pressure show a very marked primary summit, whose proportionate magnitude is modified by increase of pressure, then the tracing taken at the higher pressure more closely represents the pulse-wave. The form of trace at the lower pressure may, however, have much significance, and in these cases the whole of the information to be derived from the sphygmograph cannot be compressed into one curve, but requires at least two for its expression, namely, that trace which has the greatest amplitude, and another taken at a higher pressure.'¹

The chief points to be attended to therefore in order to get a perfect tracing are:

- (1) The accurate adjustment of the instrument, and
- (2) The proper regulation of the pressure.

The following tracings, which were taken consecutively from the same pulse, illustrate the effects of different degrees of pressure.

Speaking generally it may be said that pulses of high tension require a considerable amount, and pulses of low tension a small amount of pressure for their perfect development. But to this general rule there are some exceptions. In atheroma, for example, the amount of pressure required to obliterate the pulse is usually considerable, the arteries are abnormally full, but the condition is not necessarily one of high tension.² In cases of atheroma the development of a pointed apex is often interfered with, unless a small amount of pressure be employed.

In comparing the tracings from different arteries,—the two, radials, for example,—a procedure which is desirable in all

¹ *Journal of Anatomy and Physiology*, vol. x. p. 306.

² In the first edition of this lecture I stated that the pulse in atheroma is one of low tension. This is not always the case. I should have said, that for the perfect development of the trace, a low pressure is (as a rule) required; and that the amount of pressure required to extinguish the pulse in atheroma is not a criterion of the blood pressure, *i.e.*, the tension of the pulse, for where the vessel is rigid a considerable amount of pressure is required to overcome the resistance of the arterial wall, and it is only after the rigidity of the arterial wall is overcome that the pressure is fully exerted upon the arterial contents.

cases of supposed aneurism or solid intra-thoracic growth, the best obtainable tracing from each pulse should, in the *first* place, be taken, all the conditions (with the exception of the pressure) such as the position of the patient, the tightness with which the instrument is strapped to the wrist, etc., being so far as is possible the same; and in this connection it is very important to remember that when the heart's action becomes excited as the result of emotional or other causes, the character of the tracing may be materially modified, as is shown in figs. 73, 74, 75, and 76. *Tracings should, therefore, always be taken if possible during tranquil action of the heart.* In the *second* place, two tracings (one from either radial) should be taken with *all* the conditions, including the amount of spring pressure, the same.

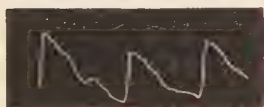


FIG. 73.—Pressure $2\frac{1}{2}$ oz.

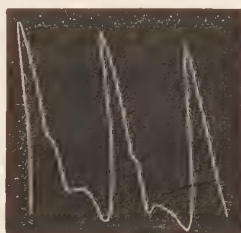


FIG. 74.—Pressure $2\frac{1}{2}$ oz.

FIGS. 73 and 74—*Alterations in the Pulse-tracing as the result of Cardiac Excitement.*—A. M., æt. 48, admitted to the Newcastle Infirmary suffering from obscure spinal symptoms. The heart became excited, and the tracing shown in fig. 74 was taken immediately after that shown in fig. 73, the instrument in the meantime remaining *in situ*. The spring pressure was the same in each case.

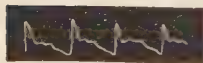


FIG. 75.—Pressure 3 oz.

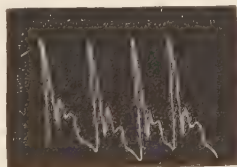


FIG. 79.—Pressure 3 oz.

FIGS. 75 and 76.—*Alterations in the Pulse-tracing which result from Cardiac Excitement.*—Figs. 75 and 76 show two tracings from a case of chlorosis. Case: E. F., æt. 19, admitted to Newcastle-on-Tyne Infirmary 3d March 1878. The tracing shown in fig. 75 was taken on 8th March; the tracing shown in fig. 76 was made three minutes later, the instrument having remained *in situ*; the heart had become excited. Pressure in both cases = 3 oz.

DIRECTIONS FOR THE APPLICATION OF DUDGEON'S
SPHYGMOGRAPH.

Dr Dudgeon gives the following directions for the application of his instrument :—

'1. Wind up the clockwork, used to drive the smoked paper along, by means of the milled button at the back of the clockwork box.

'2. Insert one end of the smoked paper (smoked side uppermost) on the right-hand side of the instrument, between the roller and small wheels.

'3. Make the patient hold out either hand open and in an easy position, the fingers pointing towards you (see fig. 77), and direct him not to move the wrist or fingers.

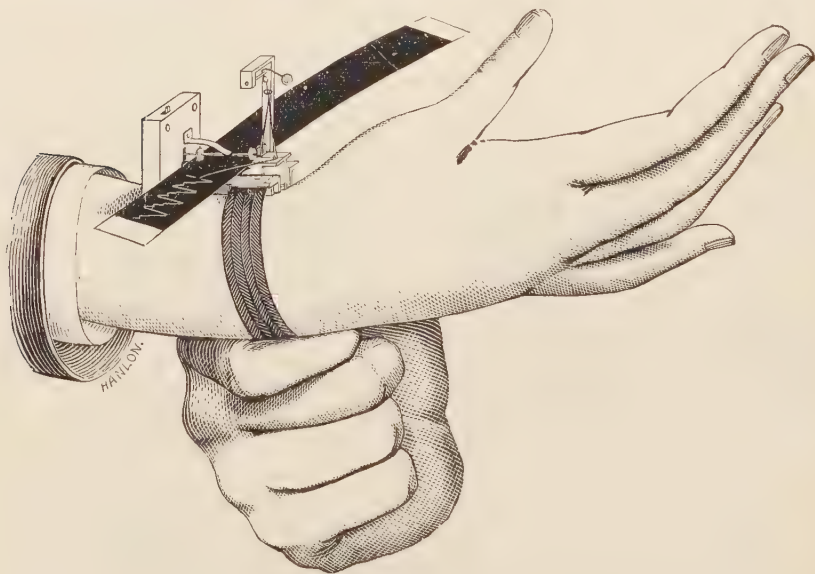


FIG. 77—*Mode of applying Dudgeon's Sphygmograph.*

'4. Ascertain the precise spot where the radial artery beats at the wrist, close behind the eminence of the os trapezium.

'5. Slip the band, the free end of which has been drawn through the clamp, over the patient's hand.

'6. Apply pressure to the spring by turning the spring-regulator so that the number of ounces, or portions of ounces, you wish, is pointed to by the indicator. The pressure may be altered at will when the instrument is fixed on the arm.

'7. Place the bulging button of the spring exactly over the artery, its long axis parallel to the course of the artery, the box containing the clockwork resting lightly on the forearm above.

'8. Retaining the instrument in its place with the left hand, draw the band through the clamp with the thumb and forefinger of the right hand, holding back the clamp with the other fingers of that hand; when the requisite tightness has been obtained, which will be known by the point of the needle working freely over the centre of the smoked paper, screw up the clamp with the left hand, so as to fix the instrument.

'9. Set the smoked paper in motion by pushing towards the right the small handle on the top of the clockwork box.

'10. Let the paper run through, and do not touch the instrument or the patient, unless to support his hand in your own right hand, to secure perfect steadiness.

'11. Catch the paper as it passes out of the instrument in your own left hand.

'12. Stop the clockwork as soon as the paper has passed.'—*The Sphygmograph*, p. 67.

Having now described the sphygmograph and its mode of application, I will next consider the characters of the normal pulse tracing, and the modifications which occur in it.

ANALYSIS OF A SPHYGMOGRAPHIC TRACING.

A pulse tracing consists of a series of curves, each one of which corresponds to one beat of the pulse, and each one of which corresponds (in time) to a complete cardiac revolution, *i.e.*, the time which elapses from the commencement of one ventricular systole to the termination of the ventricular diastole.

Each individual pulse curve, *a* to *a'* (fig. 78), may be artificially divided into the following parts:—

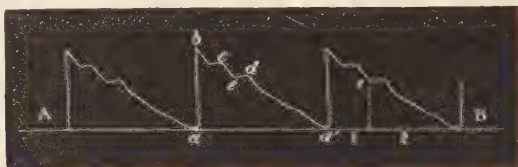
1. A line of ascent (*a* to *b*).
2. An apex (*b*).
3. A line of descent (*b* to *a'*).

This division is convenient for descriptive purposes, but a more natural division is that which separates each pulse wave into two portions (1 and 2, fig. 78) corresponding in time to the systole and diastole of the left ventricle respectively.

I will now describe each of these different parts.

The line of ascent or up-stroke¹ (*a* to *b* fig. 78) represents the sudden distension of the arterial system which is produced by the contraction of the left ventricle at the commencement of the ventricular systole, *i.e.* when the aortic segments are suddenly opened.

It is probably also partly due to the inertia of the instrument; and, in some cases, as in atheroma (where the vessels are extremely rigid), to impulse or shock.



Pressure 3 oz.

FIG. 78.—*Sphygmographic Tracing of Normal Pulse.*—Male, æt. 25, admitted to the Newcastle Infirmary suffering from psoriasis.

(1.) Line of ascent, up-stroke or percussion stroke = *a* to *b*. (2.) Apex = *b*.
(3.) Line of descent = *b* to *a'*; *d* = aortic or diastolic wave; *e* = aortic notch; *c* = tidal wave. A B = base or respiratory line. 1 = Systolic portion of the tracing, *i.e.* with reference to the systole and diastole of the ventricle, not of the artery. 2 = Diastole portion of the tracing.

(Note—Dr Mahomed tells me that he thinks the tidal wave in this tracing is rather too sustained, considering the amount of pressure, viz. 3 oz.)

The *direction* of the up-stroke (whether vertical or oblique) depends (chiefly) upon:—

1. The suddenness of the ventricular systole.
2. The condition of the aortic segments.
3. In some degree, the facility with which the blood wave is propagated from the base of the aorta to the radial artery, and
4. The condition of the arterial (radial) coats.

In the normal tracing the up-stroke is nearly vertical, for the contraction of the ventricle occurs suddenly, and there is no undue resistance in the aortic segments. When the ventricular contraction is more sudden than in

¹ It is better, I think, not to use the term *percussion stroke*, proposed by Mahomed, for unless the arteries are rendered extremely rigid by atheroma, percussion or shock probably takes little or no part in the production of the up-stroke.

health, as it is, for example, in some cases of aortic regurgitation, and in conditions of cardiac excitement, the up-stroke is quite vertical, or (in tracings taken with Marey's instrument) it may even slope backwards. (See figs. 79 and 80.)¹

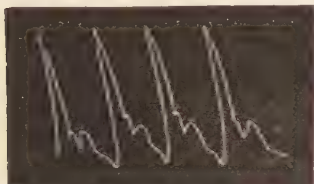


FIG. 79.—Pulse-tracing during Cardiac Excitement.

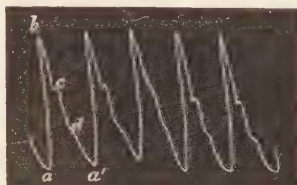


FIG. 80.—Aortic regurgitation.

Vice versâ when the ventricular contraction is slow and hesitating, as in some cases of cardiac debility; when the aortic cusps are rigid; when the arteries are obstructed either by internal or external causes, such as the pressure of a tumour, the presence of an atheromatous patch (at the orifice of the innominate in the case of the right radial for example); or, when a globular aneurismal dilatation is situated between the heart and the vessel (*i.e.* the radial), the up-stroke may be oblique. (See figs. 81 and 82.)



Pressure $1\frac{1}{2}$ oz.

FIG. 81.—*Aortic Stenosis*.—J. B., æt. 51, admitted to Newcastle Infirmary 29th November 1878, suffering from anæmia and dropsy. There was a well-marked aortic systolic murmur; the left ventricle was not hypertrophied. The pulse-tracing seems to show that the murmur was organic, and not hæmic.



Pressure $2\frac{1}{4}$ oz.

FIG. 82.—*Aneurism of left Subclavian*.—J. M., æt. 50, admitted to Newcastle Infirmary 5th September 1878; all the waves in the tracing are obliterated.

¹ In cases of hypertrophy of the left ventricle with high arterial pressure, the ventricular contraction may be laboured and prolonged in order to overcome the obstruction, but the commencement of the contraction is sudden, and the up-stroke vertical.

The *height* of the up-stroke represents the degree of distension of the vessel, and depends upon—

1. The force and (to a less degree) the suddenness with which the aortic cusps are raised, *i.e.* upon the force (and suddenness) of the contraction of the left ventricle, less the resistance offered by the aortic valve-cusps.

2. The extensibility of the arterial coats, which in its turn depends upon the condition of the arterial tunics (their elasticity or rigidity), and the state of the vaso-motor system.

3. The amount of compression which is applied to the artery, *i.e.* the spring pressure of the sphygmograph.

The up-stroke is tall in simple nervous palpitation, and in cases in which a large amount of blood is suddenly thrown into the arterial system by a hypertrophied left ventricle.

In free mitral regurgitation the left ventricle may be considerably hypertrophied, but the up-stroke is short, for in such cases the large leak at the mitral orifice prevents the distention of the arterial system. So, too, in Bright's disease and atheroma, a powerful (hypertrophied) left ventricle may fail to produce a tall up-stroke, owing to the unyielding condition of the arterial wall.

Vice versâ, the up-stroke is *short* where the left ventricle is weak or acting feebly, and in all conditions in which a small amount of blood is being pumped into the arterial system, as in aortic stenosis and mitral lesions.

The apex of the tracing, or the *primary ventricular wave*¹ (*b* in fig. 78) as it may be termed, is, in the great majority of tracings,—both normal and pathological—pointed; in fact, as I have previously remarked, a rounded apex is so extremely rare, that any tracing in which the apices are rounded should be regarded with suspicion, and should never be accepted as the best attainable (most perfect) tracing without careful readjustment of the instrument and alteration of the pressure.

¹ The term '*percussion wave*' is applied to the apex by Mahomed.

A rounded apex does, however, occasionally occur. It is met with in some aneurisms; the angles of the pulse curve are, as it were, flattened out (obliterated) either in the passage of the blood wave through the sac of the aneurism, or by alterations at the mouths of the vessels arising from the sac, or as the result of the pressure of the aneurismal sac on the vessels arising from it. A rounded apex is also seen in some cases of aortic stenosis; but in these cases, and in cases of atheroma, a pointed apex can, I think, usually be obtained by accurate adjustment of the instrument and careful regulation of the pressure.

The line of descent.—In the normal pulse tracing the line of descent (*b* to *a'* in fig. 78) is gradual,¹ and is interrupted by one or more secondary waves, the most important of which is the dicrotic.

The *direction* of the line of descent depends upon—(*a*) the facility with which the blood passes out of the arteries; (*b*) the rapidity of the heart's action; and (to a much less extent) (*c*) the condition of the arterial coats. In the normal condition of things the blood takes some time to flow from the arterial system into the capillaries, the recoil of the arteries is gradual, and the line of descent is sloping.

When the outflow from the arterial system is more difficult than in health, as for example, in the cirrhotic form of Bright's disease, the line of descent is still more gradual. *Vice versâ* when the outflow from the arterial system is very rapid in consequence of a dilated condition of the small arteries and capillaries; and in aortic regurgitation, in which the arteries suddenly collapse in consequence of the back flow through the aortic valves, the line of descent becomes more and more vertical in proportion to the freeness of the outflow and the rapidity of the action of the heart.

The *dicrotic* wave, or the *aortic systolic wave*, as it may be called, is usually present in a normal pulse tracing, and

¹ The line of descent is sloping, because the recoil of the artery is gradual. In this respect there is, therefore, a marked difference between the up-stroke and the line of descent

corresponds to that period of the cardiac cycle which immediately follows the closure of the aortic valve cusps: while the point of the tracing which immediately precedes it (*e* in fig. 78), and which is generally, but not universally,¹ believed to correspond in time to the closure of the aortic segments, is termed the aortic notch.

Where the vaso-motor tone is very good, and the arterial tension high,—as it is in some healthy persons,—the dicrotic wave is very feebly marked or altogether absent. (See fig. 83.)



FIG. 83.—*Pulse tracing of good tension (pressure 3 oz.)* The pulse is slightly irregular, but otherwise normal; the dicrotic wave is scarcely perceptible.

The exact cause of the dicrotic wave has given rise to much debate; but most physiologists are agreed that it is in great part due to the recoil current from the closed aortic valve,—an opinion which is confirmed by clinical observation. Dr Galabin, while agreeing that the great cause of the dicrotic wave is a recoil current from the aortic valves, thinks that its production is aided by the inertia of the fluid.²

Dr Roy differs from this opinion. As the result of careful experiment, he suggests that the secondary waves which appear in the unopened artery under normal blood-pressure, are the result of an active vermicular contraction of the muscular coat of the arteries. He completely rejects the theories which would ascribe these undulations to reflected,

¹ *A Text Book of Physiology*, 4th edition, by Professor Michael Foster, p. 172.

² Dr Galabin explains the mode of production of the dicrotic wave in the following manner:—‘The first cause of the dicrotic wave is that which has been very generally accepted as depending upon the aortic valves. For let us consider a section of artery close to the valves. When the influx from the heart suddenly ceases at the end of systole, the fluid for an instant continues to flow away out of the section on account of its acquired velocity, and the pressure in the section, therefore, rapidly falls, and the artery contracts. As soon as the velocity of the fluid is checked by the pressure in front, a reflux takes place, which, being stopped by the valves, causes a second increase of pressure and second expansion. This is propagated as the dicrotic wave into the periphery, and may itself again call out a second similar oscillation or tricotic wave, which is not unfrequently seen in the pulse. Even in the total absence of aortic valves, the reflux, meeting with the current entering the ventricle, may cause a second

opening and closing waves, etc., or in fact to secondary waves at all, of whatever character. The tracings which he obtained from the opened artery of a rabbit, under a normal blood-pressure, never showed 'the slightest trace of secondary waves superposed on the primary or pulse wave,' although the instrument he used was quite delicate enough to record them did they really exist.'—*Michael Foster's Journal of Physiology*, 1879-80, p. 76.

The dicrotic wave is absent or feebly marked in free aortic regurgitation. (See figs. 84 and 85.)

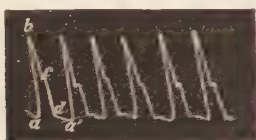


FIG. 84.—Pressure $2\frac{1}{2}$ oz.

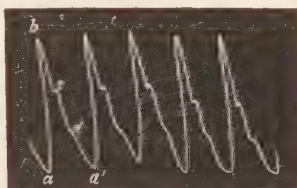


FIG. 85.—Pressure 3 oz.

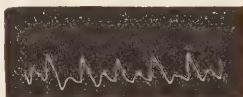
FIG. 84.—*Aortic Regurgitation*.—Case: G. A., æt. 56, admitted to Newcastle Infirmary 21st February 1878, suffering from shortness of breath and swelling of feet. Had been ill for three months. The face was pale and anxious, lips slightly dusky. Double aortic murmur; heart considerably hypertrophied; apex beat between 6th and 7th ribs, 3 inches below and 2 inches outside left nipple. Considerable hypertrophy and engorgement of right heart. Died 5th March 1878. Aorta very atheromatous; aortic valves very incompetent; segments shrunken, turned in towards the ventricle; coronary arteries much obstructed; cardiac walls fatty; left ventricle dilated; pericardium adherent. The arteries were practically empty during the ventricular diastole. *a-b*=up-stroke; *b*=apex; *c*=tidal wave; *d* indicates the position of the aortic wave, which is absent in this tracing.

FIG. 85.—*Aortic Regurgitation*.—Taken from the same patient as No. 84, after administration of digitalis. The letters have the same significance as in fig. 84.

increase of pressure or dicrotic wave, although this will be much less than in the former case. If the fluid in the tubes be air instead of blood or water, its momentum is so small that its velocity is checked instantly at the end of systole, and there is no perceptible dicrotic wave. If, on the contrary, mercury be taken, both the dicrotic and succeeding waves become enormous, on account of the great momentum of the fluid, as was shown by Marey. The fluid remaining the same, the oscillation will be more ample the greater the initial velocity, and the more slowly that velocity is checked. Thus dicrotism is promoted by a sudden action of the heart, and also by distensibility of arteries, by lowness of arterial pressure, and by freedom of outflow. I think that in considering this origin of the dicrotic wave, sufficient attention has not generally been paid to the important part played in it by the inertia of the fluid, and to the fact that the aortic valves, although extremely important, are not absolutely essential.'—*Journal of Anatomy and Physiology*, vol. x. p. 303.

It is also faintly marked in some pulses of high tension, and in some cases in which the elasticity of the arteries is much impaired, as in advanced atheroma.

It is sometimes greatly exaggerated, and the pulse is then said to be *dicrotic* (see figs. 87, 88, and 89).



Pressure 3 oz.

FIG. 87.—*Dicrotism*.—A. H., æt. 32, admitted to Newcastle Infirmary 21st March 1878, with an enormous scrofulous kidney. There were occasional rigors. This tracing was made during a rigor, the temperature being 100° F.

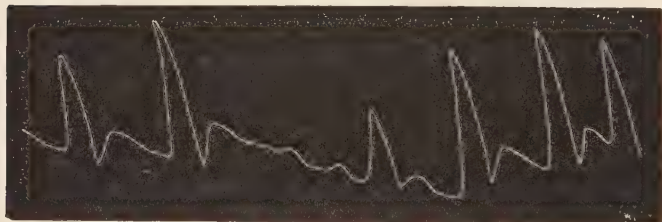


FIG. 88.—*Irregular and Dicrotic Pulse in Pneumonia*.—J. R., æt. 68, admitted to the Newcastle Infirmary 28th December 1878. Died 31st December. Croupous pneumonia, limited to the upper lobe of the right lung. No cardiac affection.

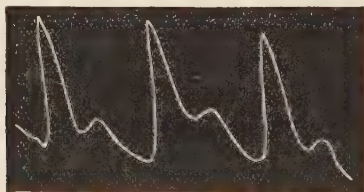


FIG. 89.—*Dicrotic Pulse from a case of Rheumatic Fever*.

Degrees of dicrotism, and their significance.—Various degrees of dicrotism occur, 'to which terms have been applied indicating the relation of what is known as the "dicrotic notch" to the respiratory line of the tracing' (Mahomed).¹

¹ *Gant's Surgery*, vol. i. p. 56.

They may be said to represent the relative condition of the artery at the point where the sphygmograph is applied, as regards its fulness or state of distention at the commencement, and at the termination of the ventricular systole respectively.

(a) When the dicrotic wave is well marked, but the aortic notch C is above the base line A B (see fig. 90), the pulse is called *dicrotic*. In this condition the artery is more distended at the end of the ventricular systole than it is at the commencement.

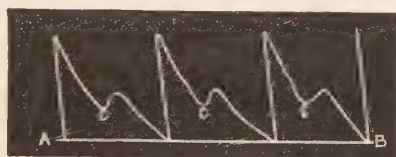


FIG. 90.—*Dicrotic Pulse.* (After Mahomed.) A B, =base line ; C, =aortic notch.

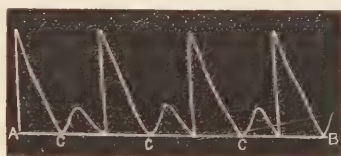


FIG. 91.—*Fully Dicrotic Pulse.* (After Mahomed.) A B, =base line ;
C, = aortic notch.

(b) When the aortic notch C reaches the level of the respiratory or base line A B, the pulse is called *fully dicrotic*. In this condition the artery is *apparently* no more distended at the end of the ventricular systole than it is at its commencement, and it is *apparently* less distended than it is during the ventricular diastole¹ (see fig. 91).

(c) When the aortic notch C sinks below the level of the

¹ This does not of course imply that the arterial system, as a whole, is more empty at the end of the ventricular systole than it is during the ventricular diastole, but simply that the vessel at the point where the observation is taken, *apparently* presents such a condition ; I say *apparently*, for the depression of the curve at the end of the ventricular systole is doubtless in part due to the sudden fall of the lever itself.

respiratory line A B, the pulse is called *hyperdicrotic*. (See fig. 92). In this condition the artery (at the point of observation) is *apparently* less distended at the termination of the ventricular systole than it is at its commencement.

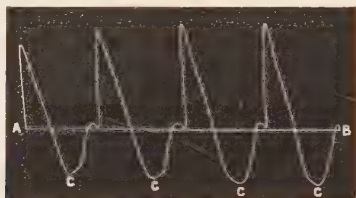


FIG. 92—*Hyperdicrotic Pulse.* (After Mahomed.) A B,=base line; C,=aortic notch.

When dicotism is well marked, *i.e.*, when the pulse is fully dicrotic, each cardiac cycle is (apparently) attended by two pulse beats, hence the term dicrotic, or double pulse.

The conditions which favour dicotism are:—

1. A low condition of arterial tension.
2. Freedom of outflow from the arterial system, *i.e.* through the capillaries.

(Both of these conditions are usually due to one and the same cause, *viz.*, loss of vaso-motor tone).

3. A sudden sharp ventricular systole.
4. Elasticity of the arterial walls.

According to Dr Burdon Sanderson, 'dicotism is characteristic of that condition of the circulation in which the arterial pressure is diminished, while the venous is increased. It denotes that the capillary current, instead of being constant in its rate of movement, is markedly accelerated during diastole, and retarded during the diastolic interval.'

Dr Roy thinks that the dicrotic wave of fever, which is associated with reduced blood-pressure, is due to a secondary and, most probably, reflected wave.

He found that any considerable reduction of the medium blood-pressure, from whatever cause, leads to the appearance of a dicrotic pulse-wave in tracings both from the opened and unopened artery.

This form of dicotism,' he says, 'must not be confounded, as is often

done, with those undulations more or less marked, which gave to the pulse curve in health its characteristic outline. The dicrotism from reduction of blood-pressure, as it is seen in tracings from the now opened artery, is characterised by the fact, that it does not disappear when the extra-arterial pressure is raised nearly as high as the blood-pressure, showing that it is really due to a secondary and most probably reflected wave.'—*Michael Foster's Journal of Physiology*, 1879, 1880, p. 80.

Clinically, the pulse is dicrotic in cases in which the nerve tone (vaso-motor system) is feeble. Many persons who apparently enjoy good health have dicrotic pulses. Such persons are easily 'knocked up,' are unable to undergo any severe and prolonged strain, and are most unfavourable subjects for an attack of continued fever; in them a severe pneumonia, or an attack of typhus, would almost certainly be fatal. A comparatively slight elevation of temperature in such persons causes the pulse to become *fully dicrotic* or even *hyperdicrotic*. (See figs. 93, 94, 95.)

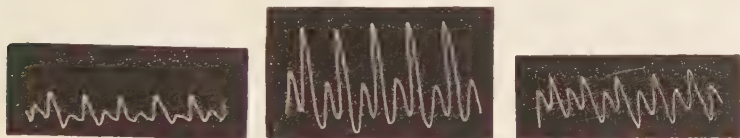


FIG. 93.—Pressure 3 oz. FIG. 94.—Pressure 3 oz. FIG. 95.—Pressure 4 oz.

FIG. 93.—*Dicrotism*.—A. H., æt. 32, admitted to Newcastle Infirmary 21st March 1878, with an enormous scrofulous kidney. There were occasional rigors. This tracing was made during a rigor, the temperature being 100° F. A draught of hot milk was administered, and the tracing, shown in fig. 94, was taken. Tracing fig. 95 a few minutes later.

FIG. 94.—*Hyperdicrotism*.

FIG. 95.—*Hyperdicrotism*.

The great clinical condition with which dicrotism is associated is pyrexia. Where a considerable elevation of temperature (102°-104° Fahr.) continues for some time, as in enteric and typhus fever for example, the pulse usually becomes dicrotic. A dicrotic pulse under such circumstances indicates the free administration of stimulants.

Hyperdicrotism is (as a rule) only seen in cases of high fever with great exhaustion, but in debilitated subjects it may, as I have previously remarked, be produced by slight

elevations of temperature. In the hyperdicrotic pulse the second (dicrotic) beat is cut short by the up-stroke of the ventricular systole of the next beat.

If the rapidity of the hyperdicrotic pulse is increased the second beat is lost altogether, and the pulse is then said to be *monocrotic*. (See fig. 96.)

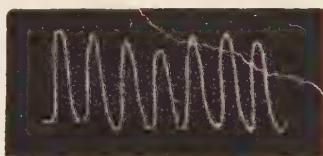


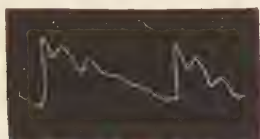
FIG. 96.—*Monocrotic Pulse.* (After Riegel.)

The *predicrotic*, *true tidal*, or *second ventricular systolic wave*.

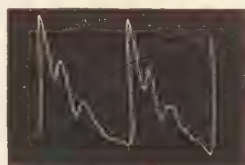
Between the apex of the tracing and the aortic notch a second wave (see fig. 78) is often, but not always, seen. Dr Mahomed thinks that it corresponds to the steady onward passage of the blood which results from the ventricular systole, hence he has termed it the *true tidal* wave. It is sometimes called the *predicrotic* wave, because of its relationship to the dicrotic wave. It may, I think, with advantage be termed the *second ventricular systolic wave*, for it occurs after the apex or first ventricular systolic wave, and during the systole of the ventricle.

The essential condition, which favours the production of the second ventricular systolic wave, is increase of the arterial tension during the ventricular systole. This condition (increased arterial tension) is usually due to difficulty of arterial outflow as in Bright's disease (see figs. 97 and 98), and in atheroma (see fig. 99); but it may also result from an excessive amount of blood being propelled into the arterial system at each ventricular systole. An excellent clinical example of the latter condition is seen in aortic regurgitation, in which a powerful and dilated ventricle propels a large quantity of blood into the arterial system, producing high tension during systole, with a well-marked *predicrotic wave*, but in which the

arterial pressure during the ventricular diastole is extremely feeble.¹



Pressure 5 oz.



Pressure 4 oz.

FIG. 97.—*Chronic Bright's Disease*.—D. G., æt. 40, admitted to Newcastle Infirmary 5th September 1878, suffering from renal dropsy (large white kidney). The pulse is one of high tension; the tidal wave is strongly marked.

FIG. 98.—*Acute Bright's Disease*.—Tidal wave strongly marked, from a patient admitted to the Newcastle Infirmary under the care of Dr Drummond.



FIG. 99.—*Atheroma and Aneurism of Aortic Arch*.—J. D., æt. 52, admitted to Newcastle Infirmary 21st February 1878, suffering from aneurism of the ascending portion of the aortic arch and atheroma. The tidal wave is very strongly marked. There was no perceptible difference between the two pulses.

Vice versâ the second ventricular systolic wave is absent in those cases in which the arterial tension during the ventricular systole is low. Now, low tension during the ventricular systole may be due either to an insufficient amount of blood being pumped into the arterial system during the ventricular contraction, a condition which is seen in cases of cardiac weakness, mitral disease, etc.; or, it may result from an abnormally free outflow from the arterial system during the ventricular systole, a condition which is

¹ Dr Galabin (formerly) supposed that the separation of the primary, or so called 'percussion' and tidal waves did not really exist in the artery, but was produced in the trace by the velocity acquired by the sphygmograph in the sudden primary up-stroke. Further observation, he states, 'has convinced him that, although this explanation applies to many cases, it yet does not express the whole truth, and that in some instances at least there is a real first secondary wave or oscillatory expansion in the artery, *i.e.* the tidal or predicrotic wave.'—*Journal of Anatomy and Physiology*, vol. x. p. 299.

due to a dilated condition of the small arteries and (?) capillaries. In such cases the pulse rapidly falls away *during the ventricular systole*, in other words, after the first distention of the arterial wall there is a quick and rapid collapse, which is only arrested by the occurrence of the dicrotic wave; and this is, as we have seen, chiefly a recoil wave from the closed aortic valves. A rapid collapse of this description is best marked in the dicrotic and hyperdicrotic pulse of fever, and in conditions of vaso-motor debility and relaxed vessels. Hence it will be easily understood why in these cases the second ventricular systolic wave is not present.

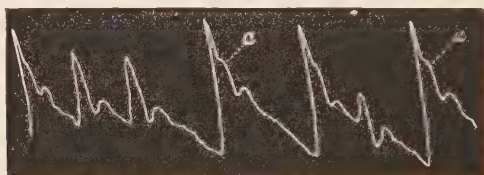
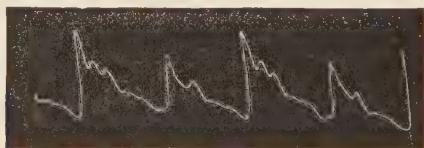


FIG. 100.—*Irregular Pulse in mitral regurgitation, with hypertrophy of the Left Ventricle.*—In the tallest curves the tidal wave, *a*, is well-marked, while it is absent in the smaller ones. The letters *a, a*, point to the predicrotic wave.



Pressure $4\frac{1}{4}$ oz.

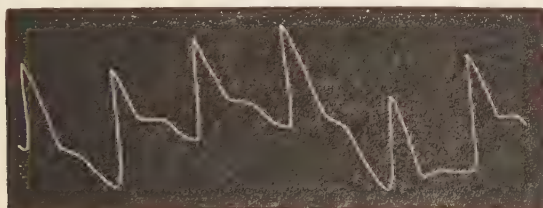
FIG. 101.—*General Atheroma.*—J. D., æt. 60, admitted to Newcastle Infirmary 21st October 1878. The radials were very tortuous and rigid; there was no valvular lesion. The tidal wave only occurs every alternate beat.

It not unfrequently happens that the predicrotic wave is present in some pulse curves of a tracing, but absent in others. This condition, which depends of course upon the fact that the arterial pressure during the ventricular systole is greater during some pulsations than during others, is especially frequent in mitral stenosis, in which condition a varying amount of blood is apt to be discharged into the cavity of the left ventricle, and thence into the arterial system. (See fig. 100.) Occasionally the predicrotic wave occurs every alternate beat, as shown in fig. 101; in that case the irregularity was probably due to nervous causes.

Other secondary waves sometimes occur in the lower part of the line of descent. Their exact cause is obscure, but so far as is at present known, they are of little practical importance.

These waves are probably as a rule due to the inertia of the instrument. Occasionally a small wave is seen to occur immediately before the up-stroke, *i.e.* immediately before the contraction of the ventricle. Possibly it may be due to the contraction of the left auricle.

Respiratory or base line.—In a normal tracing the lowest points of the up-strokes of succeeding pulse waves are on the same horizontal plane (see fig. 78), and a line drawn through the bases of the up-strokes is called the *base* or *respiratory* line. The latter term (respiratory line) is applied to the base line because inspiration and expiration exert, sometimes even in health, but notably in some cases of disease, a marked influence upon it. During a full and sudden inspiration the arterial tension is lowered, and the base line falls; during expiration, on the contrary, the arterial tension is increased, and the base line rises. In cases of spasmodic asthma and severe dyspnoea the base line may be very uneven. (See figs. 101, 102.)



Pressure $3\frac{3}{4}$ oz.

FIG. 102.—*Uneven Respiratory Line.*—J. R., æt. 31, admitted to Newcastle Infirmary 26th December 1878, suffering from acute bronchitis.

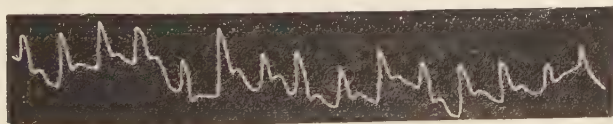


FIG. 103.—*Uneven Respiratory Line (from a case of Spasmodic Asthma).*
The tracing was taken during the paroxysm.

In the normal pulse-tracing, during ordinary (tranquil) respiration, each consecutive pulse curve is an exact repetition of the preceding one; in other words, the pulse is regular in time, the different pulse waves are equal in volume, and the individual character of succeeding pulse curves, in respect to the different parts of which they are composed, is the same. When the pulse-tension is low, or when the respiration is active, succeeding pulse curves are seldom quite identical, but are more or less modified by inspiration and expiration, as described above.

Having analysed the normal pulse tracing, and described the more important modifications which it undergoes, I will next proceed to consider the character of the pulse in health and disease.

THE FREQUENCY OF THE PULSE.

The frequency of the pulse is determined by counting the number of pulsations which occur in a minute. To insure accuracy, the pulse should be counted for a whole minute, and not for a quarter of a minute, as is sometimes done. In some cases in which the pulse at the wrist is very feeble, the frequency of the cardiac contractions can be best determined by auscultation over the præcordia.

The exact frequency of the pulse can also be determined by means of the sphygmograph. Mahomed's modification of Marey's instrument is so constructed that four inches of the slide are propelled past the point of the writing lever in ten seconds. In order, therefore, to ascertain the frequency of the pulse per minute, the number of pulse waves in four inches of the tracing must be multiplied by six. Dudgeon's instrument is also constructed, so that six times the number of pulsations, traced on the slip of paper, give the number of beats per minute.¹

In counting a very rapid pulse, Dr Abbot describes, in the *New York Medical Record* for August 12, 1882 (quoted in the *Medical Times and Gazette*, September 30, 1882), a method which he adopted for counting the heart's action during some experiments he performed with alcohol on birds. He found that he was unable to count by the usual mode

¹ These measurements only hold good so long as the slide is travelling at full speed. To insure accuracy, therefore, the clock-work should be fully wound up before the tracing is taken.

when the cardiac contractions exceeded 240 per minute, whereas, by the method he now describes, he easily counted 280. 'During a definite part of a minute, usually one-fourth, dots were made with a lead pencil upon a sheet of paper, *synchronous with the heart's beats*, as heard over the cardiac region. The dots were then counted. A pulse of *four hundred* could be taken in this way, provided each pulsation were distinct enough to be discriminated by the touch. The indistinctness of the separate pulsations alone fixes the limits to the use of this method, as the human hand is capable of making intelligently and with accuracy at the rate of 450 dots per minute, for thirty seconds, which rate is probably beyond not only that of the human heart, but also of the pulse of any of the lower animals available for experiment. I have had a sufficient experience with this method,' he says, 'to know that it is of practical value, especially with children. All movements, whether of the body or not, that can be seen, felt, or heard, can be thus counted up to 400 or 500 per minute, provided that they are sufficiently distinct to be discriminated.'

Frequency in health.—The normal frequency varies in different individuals, and in the same individual under different circumstances. The average normal rate in the adult male in a state of rest is 72, but there are many exceptions. In some persons the pulse rate is habitually as high as 100, in others as low as 50. In practice, therefore, such idiosyncracies must be kept in view. The pulse is quicker in children than in adults, but it quickens slightly again in old age; it is quicker in women than in men. The pulse rate is increased by active exercise (bodily or mental). It varies, too, with the position of the body, being quicker in the standing than in the sitting, and in the sitting than in the recumbent position. Its frequency also varies with the time of day, being lower in the early morning hours. It is decreased during sleep and increased after a meal. It varies with the temperature of the body, and is to a slight extent influenced by the atmospheric pressure. The frequency of the pulse is also profoundly influenced by emotional disturbances, and by the mental condition, hence it is often difficult to get a proper estimate of the pulse-rate in children and nervous persons, the mere presence of the doctor being sufficient to increase the frequency by 10, 20, 30, or even 40 beats. Due allowance must of course be made for this and other disturbing

causes. It is often a good plan to count the pulse at the end of the visit, or at all events to allow sufficient time for any temporary alteration in the pulse-rate to have disappeared.

TABLE OF THE AVERAGE PULSE RATE AT DIFFERENT AGES.

Fœtus in utero,	. . .	about 140
Child newly born,	. . .	140—135
„ 1st year,	. . .	120—110
„ 2d year,	. . .	105—100
„ 3d year,	. . .	100—85
7th—14th year,	. . .	85—80
14th—20th year,	. . .	80—72
21st—60th year,	. . .	70—75
Old age,	. . .	75—80

Alterations in the pulse-rate, which occur in disease.

The pulse-rate may be either increased or diminished by disease.

The pulse-rate is increased in:—

1. *Pyrexia* (increased temperature).—As a general rule the amount of increase varies with the height of the temperature. According to Dr Aitken, an increase of temperature of one degree above 98° Fahr. corresponds with an increase of ten beats of the pulse per minute, as shown in the following table:—

Temp. Fahr.	Pulse-rate.	Temp. Fahr.	Pulse-rate.
98° . .	60	103° . .	110
99° . .	70	104° . .	120
100° . .	80	105° . .	130
101° . .	90	106° . .	140
102° . .	100		

Exceptions.—In some cases of typhoid, especially in its earlier stages, and in meningitis, the pulse may be slower than natural. At the commencement, too, of some cases of pericarditis the frequency of the pulse is diminished—(Stokes).

2. *Conditions associated with extreme debility.*—This is chiefly the case where there has been some previous elevation of temperature, or where the nerve irritability of the heart is increased.

3. *Cases in which the vagus is paralysed or the cervical sympathetic irritated*—In these cases the nerve balance of the heart is deranged, and the pulse-rate increased. In a few cases (as for instance, in exophthalmic goitre in which the sympathetic is irritated, and in the later stages of basilar meningitis, in which there is probably paralysis of the vagus), the nerve derangement depends upon organic disease; but in the large majority of cases, as in hysteria, in which affection the pulse frequency may be enormously increased, the condition is a functional one.

4. *Some cases of organic cardiac disease*, especially mitral regurgitation (see fig. 104), and (to a less extent) in aortic regurgitation (see fig. 105).



Pressure 3 oz.

FIG. 104.—*Mitral Regurgitation*.—M. A. C., æt. 16, admitted to Newcastle Infirmary 24th January 1878, suffering from cough and shortness of breath, dating from an attack of rheumatic fever two months previously. Heart's action very rapid (120-130). The first sound appeared to be reduplicated; a systolic murmur was audible at the apex when the heart became slower.

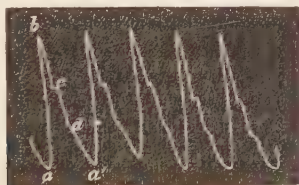


FIG. 105.—*Pulse tracing from a Case of Aortic Regurgitation*.

The Clinical Significance of Increased Frequency of the Pulse.

Increased frequency of the pulse, when not merely temporary, is very suggestive of pyrexia; it is, however, *per se*, an unreliable sign of fever, for, on the one hand, fever may be present without any increase (a diminished pulse-rate being, as I have already pointed out, sometimes met with, as in the earlier stages of typhoid and in meningitis); and, on the other, increased frequency of the pulse may occur without

fever, or even with a low temperature, as in pro-agonistic collapse. In order to ascertain the cause of the increased frequency of the pulse, the first step is to take the temperature. If there is no pyrexia, the causes mentioned under heads 2, 3, 4 must be looked for.

A slow pulse may be due to—

1. *Functional derangement of the heart.*—This is probably the cause of the slow pulse which occurs in jaundice, in some cases of gout, etc.

Non-febrile jaundice usually produces a retarded action of the heart and diminished arterial tension. The pulse may fall to 50, 40, or even 20 beats in the minute, and it may also be irregular.

Dr Wickham Legg and others believe, that the slowness of the pulse is produced by the presence of unchanged biliary acids in the blood.

Dr Murchison has seen a slow pulse (36-60 per minute) in cases of hepatic disease in which there was no jaundice.

2. *Organic lesions of the heart*, such as fatty degeneration of the left ventricle and aortic stenosis. In the former case (weakness of the ventricular wall) a slow pulse is perhaps exceptional, and is only seen when the patient is at rest. Any exertion which throws a strain on the beat, is attended in these cases with a quick, rather than with a slow, pulse. In the latter case (aortic stenosis) the left ventricle has difficulty in emptying itself, its contraction is prolonged and somewhat laboured, and the frequency of the pulse is diminished.

3. *Lesions of the nervous system*, in which the cardio-inhibitory centre in the medulla or the branches of the vagus which pass from that centre to the heart are irritated (stimulated). In some cases the condition is a temporary (functional) one, as for example, in those cases in which a slow pulse is associated with a neuralgic headache (megrim); in others, as for example meningitis (in the earlier stages of which the pulse may be abnormally slow), the lesion is organic.

4. *The rapid defervescence of fever*; the pulse-rate may in these cases rapidly fall from a high rate to a point much below the normal.

5. *Reflex stimulation of the cardio-inhibitory centre in the medulla.*—The reflex impulse may in all probability be generated by powerful stimulation of any peripheral nerve ; but it most frequently arises in the alimentary tract.

The slow pulse which is so frequently seen in the earlier stages of typhoid, is probably due to reflex inhibition of the heart—the local lesion of the intestine stimulates the mesenteric nerves, producing an impulse which travels to the medulla, and is reflected down the vagus to the heart.

The Clinical Significance of a Slow Pulse.

A slow pulse *per se* (*i.e.* without any associated signs or symptoms) is of little practical importance, the most frequent cause (provided that it does not depend upon idiosyncrasy) is a temporary functional derangement of the heart. The possibility of the condition being due to irritation of the vagi must be remembered, and the symptoms of disease at the base of the brain or in the course of the vagus looked for. In other cases (*i.e.* where there are associated signs and symptoms) the clinical significance of a slow pulse entirely depends upon the cause of the condition, and the prognosis must be guided accordingly.

Variability of the pulse-rate.

The pulse-rate is in some cases liable to very marked fluctuations. In the convalescent stage of fevers, for example, and in conditions of debility, the pulse-rate is very variable ; these changes are readily produced by any trivial movement or mental excitement. I have noticed the same liability to rapid changes in the pulse-rate in the earlier stages of some cerebral cases. Extreme variability of the pulse-rate has also been noticed after concussion of the spine.¹

¹ Dr Guinoiseau, quoted in the *Medical Times and Gazette*, relates in the *Bulletin de Thérapeutique*, February 28th 1882, the case of a man who had received a concussion of the spine from a fall from a carriage on May 9th, 1881. He recovered, and was able to resume his occupation, which was laborious ; but a peculiarity in his pulse remained. Examined on October 8th, it was found that his pulse was 49 when recumbent, 73 when seated, and 109 when standing ; and on November 1st the pulse-rate in these positions was respectively 45, 57, and 77.

THE RHYTHM OF THE PULSE.

The normal pulse (during tranquil respiration) is perfectly regular (the individual pulse waves being of the same duration and volume, and presenting the same features as regards individual curves); but departures from the normal are common, indeed in some persons who enjoy perfect health, the pulse is habitually irregular. Such idiosyncracies are more common in old than in young people.

The alterations in rhythm which occur in disease can of course be observed by the finger, but are best studied by means of the sphygmograph. They may consist of alterations in time, alterations in volume, or differences in the sphygmographic characters of the individual pulse curves.

Time irregularities.—All degrees of time irregularity are met with. In some, the alteration is only occasional, occurring every ten, twenty, or thirty beats; in others, the normal rhythm of the pulse is very much altered or entirely lost. (See fig. 106.)

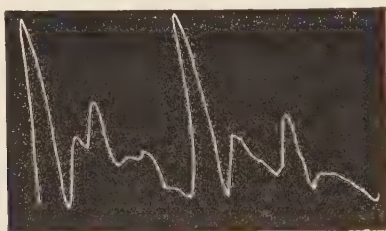


FIG. 106.—*Extreme irregularity of the pulse from a Case of Pneumonia.* Five pulse waves are shown in the tracing, which was taken twenty-four hours before death.

The irregularity sometimes consists in the omission of a beat (see figs. 107, 108); the pulse is then said to be '*intermittent*.' Intermission of the pulse may be due either to arrest of the contraction of the left ventricle—a condition which is not uncommon as the result of simple nervous derangement, and is then of comparatively little importance; or, it may be owing to the fact that some of the ventricular

contractions fail (are too feeble) to raise the aortic valves and send into the arteries a pulse wave of sufficient strength to be felt at the wrist.

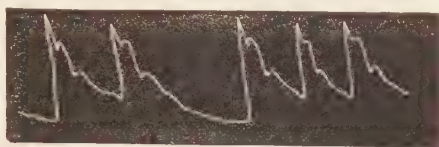
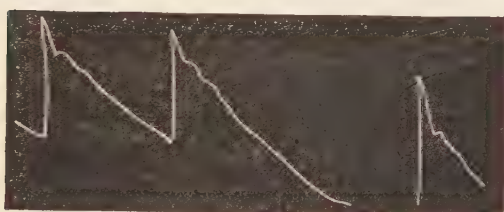


FIG. 107.—*Intermittent Pulse.*



Pressure 4 oz.

FIG. 108.—*Intermittent Pulse.*—J. B., æt. 38, admitted to the Newcastle Infirmary 11th July 1878, suffering from mitral regurgitation. The heart was much hypertrophied. Tracing made 6th January, when patient was much improved and attending as an out-patient.

This latter condition, which is a very serious indication of heart-failure, is chiefly met with in those cases in which the heart-walls are degenerated (dilatation, fatty and fibroid degeneration), but is also seen in some mitral lesions.

M Marey, quoted by Mahomed, *Guy's Hospital Reports*, 1879, p. 397, gives the following explanation of this condition :—‘ In a heart in which mitral regurgitation occurs, blood is forced out of the ventricle in two directions during systole ; one portion passes through the aortic orifice, the other is driven backward into the auricle. Now, when the heart is dilated, it sometimes occurs that the ventricular contraction is not of sufficient strength to overcome the arterial resistance and raise the aortic valves ; it finds it easier to force all the blood backwards through the incompetent mitral, which thus plays the part of a safety valve. Meantime this intermission, which is caused in the pulse, allows time for more blood to flow out of the arterial system through the capillaries, and when the next contraction of the heart occurs, it finds the arterial pressure considerably decreased, and it is now able to open the valves and cause another pulse-wave to pass through the arteries.’

In some cases the irregularities occur at fixed intervals, *i.e.* every two, three, or four beats. One of the most interesting of these is the *pulsus bigeminus* of Traube, in which the pulse waves run in pairs. (See figs. 109 and 110.) In other cases three pulsations occur together in a group, constituting the so called *pulsus trigeminus*. (See fig. 111.)

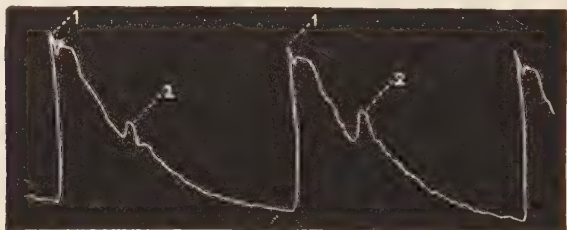


FIG. 109.—*Pulsus bigeminus*.

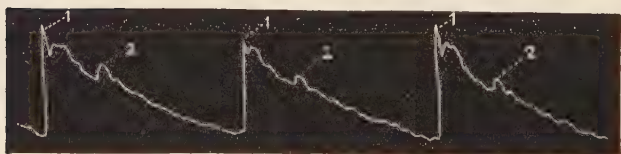


FIG. 110.—*Pulsus bigeminus*.

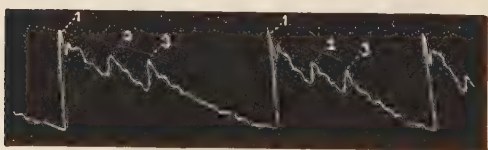


FIG. 111.—*Pulsus trigeminus*.

Irregularity in volume.—Irregularities in volume (see fig. 112) depend upon the fact that unequal quantities of blood are discharged into the arterial system at different contractions of the left ventricle. The condition is usually associated with irregularity in time, for when the time between the ventricular contractions varies, the amount of

blood which the left ventricle has to discharge will be apt to vary too.

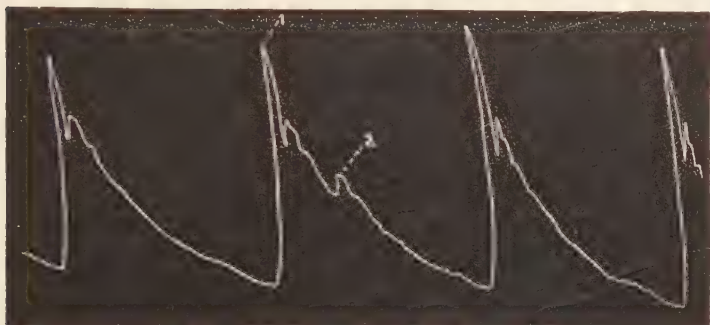
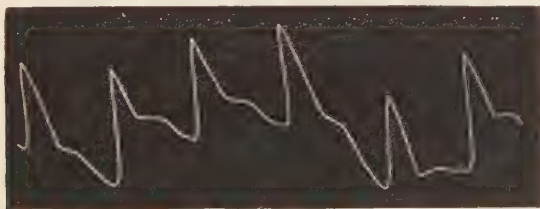


FIG. 112.

Differences in the sphygmographic characters of individual pulse waves depend upon differences in arterial tension, which in their turn may be due either to—

1. Different quantities of blood being propelled during successive contractions of the left ventricle into the arterial system, the causes of which condition I have already considered.



Pressure $3\frac{3}{4}$ oz.

FIG. 113.—*Uneven Respiratory Line.*—J. R., æt. 31, admitted to Newcastle Infirmary 26th December 1878, suffering from acute bronchitis.

2. The effects of respiration. During tranquil respiration the *respiratory* or *base line* is, as I have previously stated, a straight line. During deep inspiration and expiration, even in health, and in many pathological conditions in which the respiratory movements are profoundly affected, the 'base line' becomes very uneven (see figs. 113 and 114), and the sphygmographic character of successive pulse waves is

different; expiration increases the pulse tension and lessens the frequency of the pulse; inspiration lowers the tension and increases the frequency.¹

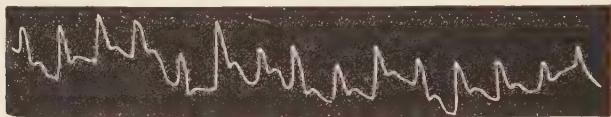


FIG. 114.—*Uneven Respiratory line (from a Case of Spasmodic Asthma).*

The tracing was taken during the paroxysm.

In extreme cases the pulse-wave may be entirely absent during inspiration. This is the *pulsus paradoxicus* of Kussmaul. The most striking examples of this condition are seen where fibrous adhesions pass between the thoracic parietes and the roots of the aorta and great vessels. During a full inspiration these fibrous bands are stretched, the vessels are constricted, and the pulse-wave is unable to reach the wrist.

The *pulsus paradoxicus* has also been noted in cases of pericarditis without constricting adhesions; also in cases of stenosis of the air-passages.

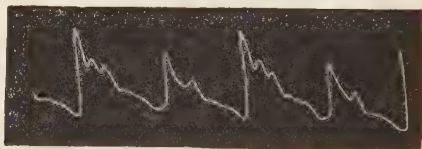


FIG. 115.—*General Atheroma.*—J. D., æt 60, admitted to Newcastle Infirmary 21st October 1878 The radials were very tortuous and rigid; there was no valvular lesion The tidal wave only occurs every alternate beat.

3. Differences in the resistance which the blood stream meets with in the peripheral vessels. This cause is probably rare, but it is difficult to account for such a pulse tracing

¹ At the beginning of inspiration the arterial pressure falls; it soon, however, begins to rise, but does not reach the maximum until some time after expiration has begun. The fall continues during the remainder of expiration, and passes on into the succeeding inspiration (Foster, page 344). In speaking of the cause of the respiratory undulations, Foster says, 'We may conclude then, that the respiratory undulations of blood-pressure are of complex origin, being partly the mechanical results of the thoracic movements, possibly also produced by the alternate expansion and collapse of the pulmonary alveoli. but probably, in addition, brought about by a rhythmical variation of the vascular peripheral resistance, the result of a rhythmical activity of the vaso-motor centre.'—Third edition, p. 348.

as that shown in fig. 115), unless we suppose either a rhythmical difference in the strength of the successive cardiac contractions, or a rhythmical difference in the peripheral resistance; in both cases the primary cause is evidently nervous, and I see no reason why a rhythmical alteration in the peripheral resistance due to vaso-motor causes might not occur.

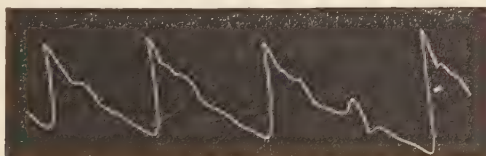
The clinical significance of inequalities of rhythm depends entirely upon the cause of the condition. Occurring *per se*, without any associated signs and symptoms of disease, the condition is of no practical importance. When there are other signs and symptoms the prognosis depends entirely upon their cause.

The chief pathological conditions associated with irregularities in time and volume are—

1. Functional derangements of the heart, such as are produced by hysterical conditions, venereal excess, gout, tobacco, tea, etc.

Physiologists have shown that when the excised heart is fed with rabbit's serum its action is apt to become intermittent. This intermittence is possibly due to the chemical action of the serum. 'Various chemical substances in the blood (natural or morbid),' says Michael Foster, 'may thus affect the heart's beat, by acting on its muscular fibres, its reflex or automatic ganglia, or its intrinsic inhibitory apparatus.'¹

2. Mitral lesions, both stenosis and regurgitation, especially after compensation has failed. (See figs. 116, 117, 118 and 119.)



Pressure $3\frac{3}{4}$ oz.

FIG. 116.—*Irregularity of the pulse.*—W. M., æt. 50, admitted to Newcastle Infirmary 30th November 1878, suffering from the usual symptoms of mitral disease. The heart's action was extremely irregular. The left ventricle was much hypertrophied. There was no rheumatic history. The symptoms were of two months' duration.

¹ *A Text-Book of Physiology*, third edition, p. 178.

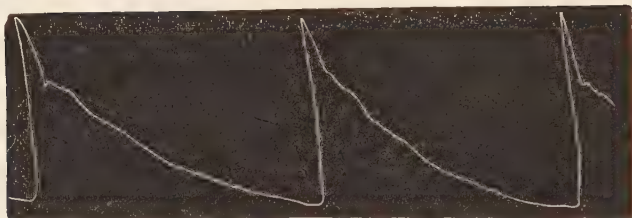
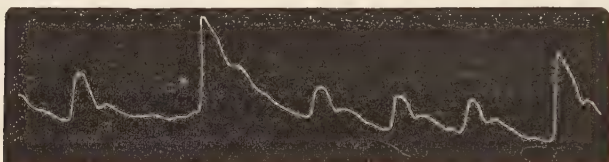


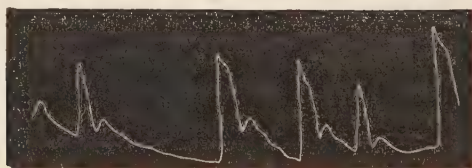
FIG. 117.

FIGS. 116 and 117 were two consecutive tracings taken on 10th December, after the patient had improved under digitalis. The intermittent action of the heart is well shown in fig. 117



Pressure 4 oz

FIG. 118 —*Mitral Regurgitation*.—S B, æt. 58, admitted to Newcastle Infirmary 25th February 1878, suffering from cardiac dropsy. There was a well-marked mitral systolic murmur, which disappeared under treatment. The heart was considerably enlarged (hypertrophied and dilated).



Pressure 3 oz

FIG. 119.—*Irregular and Intermittent Pulse*.—O. M, æt. 40, admitted to the Newcastle Infirmary suffering from cardiac dropsy. The heart was very much enlarged; apex beat $4\frac{1}{2}$ inches below and 3 inches outside left nipple; systolic mitral murmur.

There has been a good deal of debate with regard to the rhythm of the pulse in mitral stenosis. The majority of observers are, however, agreed—and with their opinion I entirely concur—that the pulse is irregular. Dr Sansom—than whom no better authority could be quoted—says, ‘We may conclude, therefore, that a pulse tracing which shows irregularity in the diastolic periods, sometimes missed pulsations, and, as described by Dr B. Foster, the occasional appearance of a small abortive pulsation in the line of descent, is very strong evidence of the existence of mitral stenosis.’—(*Diagnosis of Diseases of the Heart*, p. 269.) Dr Mahomed, who was, I believe, the first to describe this peculiar rhythm as characteristic of mitral stenosis, has since somewhat modified his views.

He says, 'Although it is very commonly present in this disease, perhaps more commonly than in any other valvular lesion, nevertheless it is not so much a sign of stenosis of the valves as of dilatation of the ventricle. It is true, that in the typical cases of mitral stenosis the ventricle is not dilated, but I am unable to say whether this irregularity only occurs in cases in which dilatation exists.'—*Guy's Hospital Reports*, 1879, p. 401.

3. Degenerations of the cardiac walls. In cases of fatty and fibroid degeneration, and, in fact, in all conditions in which there is much degeneration of the left ventricle, the heart's action may be very irregular.

4. Some affections of the central nervous system, such as meningitis, in which alterations in the condition of the intracranial circulation, such as result from sudden changes in position, etc., may cause alterations in the rhythm as well as in the rate of the heart's contractions.

THE VOLUME OF THE PULSE.

The *volume* of the pulse, which depends upon—(1) the size of the artery (radial, carotid, etc.) which is being examined; (2) the amount of blood which is propelled into the artery at each ventricular systole; and (3) the tonicity of the arterial wall, *i.e.* the condition of the vaso-motor apparatus,—may be appreciated by the finger, but is accurately measured by means of the sphygmograph.

In *health* the volume of the pulse varies from time to time, and is of course different in different individuals. There are also many modifications in disease.

A *large* pulse, *i.e.* a pulse of large volume, is seen in the following conditions:—

1. In many cases of fever during the earlier¹ periods of the attack, when the heart is acting powerfully, and propelling a large quantity of blood into vessels, the tonicity of which is already somewhat relaxed.²

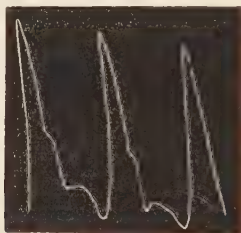
¹ During the stage of rigor the pulse is small.

² Towards the termination of cases of fever, *i.e.* after the condition has continued for some time, the pulse vessels become still more relaxed, and the pulse becomes small. It is often under these circumstances dicrotic.

2. In atheroma, when the elasticity of the arterial walls is impaired and the vessels are dilated.

3. In some cases of hypertrophy, and in some cases of simple cardiac excitement. (See figs. 120 and 121.)

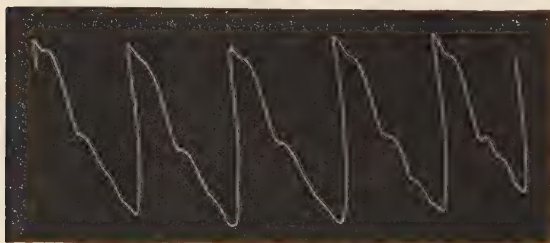
4. In aortic regurgitation the systolic portion of the tracing is of large volume, but the diastolic extremely small.



Pressure $2\frac{1}{2}$ oz.

FIG. 120 — *Alterations in the Pulse-tracings as the result of Cardiac Excitement.*—

A. M., æt 48, admitted to the Newcastle Infirmary suffering from obscure spinal symptoms. The heart became excited, and the tracing shown in fig. 120 was taken immediately after that shown in fig. 73, the instrument in the meantime remaining *in situ*. The spring pressure was the same in each case



Pressure $3\frac{3}{4}$ oz.

FIG. 121 — *Hypertrophy of Left Ventricle*—A. T., æt. 54, admitted to Newcastle Infirmary suffering from cirrhotic kidney.

A *small* pulse is met with :

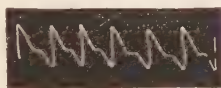
1. In those conditions in which the amount of blood discharged by the left ventricle is below the normal amount. Under this head are comprised :—

- (a) Cases (such as inanition) in which the total amount of blood in the body is reduced in quantity. (See fig. 122.)
- (b) Cases of mitral disease, more especially regurgitation and stenosis after the failure of compensation. (See fig. 123.) In the former (mitral stenosis) the left ventricle does not receive the usual (normal) amount of blood from the auricle; in the latter case, some of the blood which ought to be discharged into the aorta flows back into the auricle through the incompetent valve.



Pressure 2 oz.

FIG. 122.—*Small Weak Pulse.*—J. M., æt. 18, a soldier, admitted to the Newcastle Infirmary 23d January 1879, suffering from abscess of the liver and pericarditis. The heart was displaced upwards and to the left.



Pressure 3 oz.

FIG. 123.—*Mitral Regurgitation.*—M. A. C., æt. 16, admitted to Newcastle Infirmary 24th January 1878, suffering from cough and shortness of breath, dating from an attack of rheumatic fever two months previously. Heart's action very rapid (120-130). The first sound appeared to be reduplicated; a systolic murmur at the apex, audible when the heart became slower.

- (c) Cases of aortic stenosis (see fig. 124); the size of the pulse being in proportion to the narrowing of the orifice.



Pressure 1½ oz.

FIG. 124.—*Aortic Stenosis.*—J. B., æt. 51, admitted to Newcastle Infirmary 29th November 1878, suffering from anæmia and dropsy. There was a well-marked aortic systolic murmur; the left ventricle was not hypertrophied. The pulse-tracing seems to show that the murmur was organic.

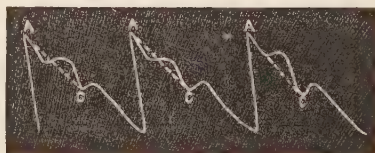
(d) Conditions of cardiac debility, both temporary and permanent. (See fig. 122) Conditions of collapse are good examples of the former ; the pulse in collapse being small and thready, while fatty and fibroid degeneration of the left ventricle, with dilatation, are types of the latter.

2 In those cases in which the vessels are unduly contracted, as in peritonitis, the cirrhotic form of kidney disease, and the cold stage of fevers, *i.e.* during the rigor.

THE COMPRESSIBILITY OR STRENGTH OF THE PULSE is a point of great practical importance, for in many cases it indicates the condition of the vaso-motor nerve apparatus, and hence of the general tone of the system.

The strength of the pulse is measured by the finger or by means of the sphygmograph, the amount of pressure required to obliterate the pulse-wave being (provided the arterial walls are healthy) the true indication of the pulse-strength.¹

Dr Mahomed gauges high tension in the following manner:—‘A line must be drawn from the apex of the up-stroke to the bottom of the notch preceding the dicrotic wave (fig. 125, A C). No part of the tracing should rise above this line ;



(After Mahomed.)

FIG. 125.—Mahomed's method of gauging high tension. The tidal wave rises above the dotted line drawn from the apex A, to the aortic notch C.

if it does, then the pulse is one of high pressure. The height of this notch is another good gauge of pressure ; the higher it is from the base line of the tracing the higher is the pressure, the nearer it approaches the line the lower is the pressure.

¹ When the arterial walls are inelastic and rigid, as in cases of atheroma, the amount of pressure required to obliterate the pulse is not a true criterion of its strength, for a certain (often a considerable) amount of pressure must be applied before any influence is exerted upon the blood wave itself.

Lastly, the duration of systole compared to that of diastole may perhaps be reckoned an important sign.' Dr Mahomed believes 'that there is a normal length of systole for a pulse of a given frequency, and that the length of the systole is increased if the arterial pressure is increased.'¹

I am in the habit of considering a pulse of high tension as synonymous with a strong pulse, and *vice versâ* a pulse of low tension with a weak pulse.²

In vigorous health the strength or tension of the pulse is considerable, but there are great differences, even in persons who present all the external appearances of good health; while alterations in the tension of the pulse are common in disease.

A pulse of *high tension* may be either large or small, the pulse of chronic Bright's disease (especially the cirrhotic form), in which the heart is notably hypertrophied, is illustrative of the former; the pulse of peritonitis is an excellent example of the latter.

A *weak* pulse, or pulse of *low tension*, is also very common in disease, and is also of great importance. A pulse of low tension is usually associated with feeble action of the heart and a relaxed condition of the blood vessels. It may be either small or large; the former—a small pulse of low tension—is seen in mitral lesions and towards the terminal period of fever; the latter—a large pulse of low tension—is also seen in some cases of fever (as for instance in certain stages of rheumatic fever), and in many persons who enjoy apparent good health, but in whom the vaso-motor tone is below par.

As a general rule, a slow pulse is a pulse of higher tension than a quick pulse, and *vice versâ*, but such a relationship is by no means constant or necessary.

¹ *Guy's Hospital Reports*, 1879, p. 371.

² I, therefore, differ from Dr Mahomed, who considers that hardness or incompressibility is the least constant character of the high pressure pulse. 'It is not unfrequent,' he says, 'to find *overfull* vessels associated with a weak or failing heart, the pulse is then often small and feeble, it is very easily compressed, and is described as a small weak pulse, which is thought usually to require stimulants; the reverse, however, is the case; bleeding or purging will be well borne by such patients, and the result will be most satisfactory.'

The clinical significance of the tension of the pulse.—The tension of the pulse is of great importance, both for diagnosis, prognosis, and treatment. Dr Mahomed, for example, has shown that in many cases of chronic Bright's disease there is in all probability a stage of the disease in which the urine is healthy (free from albumen, casts, etc.), but in which there is persistent high arterial tension, and further, that by reducing this condition of high tension the subsequent structural affection of the kidney may be prevented. So, too, a *hard* pulse associated with cardiac pain (angina pectoris) indicates a serious condition, and urgently calls for treatment.

On the other hand, a pulse of low tension is no less important both for prognosis and treatment. Persons whose vaso-motor tone is below par, *i.e.* who have weak pulses, bear severe (especially acute) disease badly; while a weak, and especially a dicrotic pulse in a case of fever, requires the free use of stimulants.

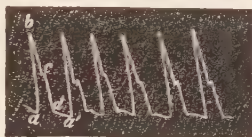
THE SPHYGMOGRAPHIC CHARACTERS OF THE INDIVIDUAL PULSE-WAVES.—Most of these points, which are of considerable practical importance, such as dicrotism, have been already fully considered; the following must also be observed:—

(1) *The relative duration of those portions of the tracing which correspond to the ventricular systole and diastole.*—In the normal condition the ventricular systole occupies, as we have previously seen, about $\frac{4}{10}$ of the entire cardiac revolution. This relationship is altered in certain cases of disease. When the heart is acting very rapidly, the diastole is relatively more reduced than the systole, and the proportional duration of the systolic to the diastolic portion of the tracing is increased. Increased temperature of the blood, has, as Dr Paul Chapman¹ has shown, a distinct influence in shortening the duration of the ventricular systole; and the same authority has observed, on coming out of a Turkish bath, 'that although

¹ *British Medical Journal*, August 19, 1882, p. 300.

the pulse-frequency may actually increase, the systole lengthens as the patient cools.' So, too, in aortic regurgitation, the systolic portion of the tracing is usually longer than the diastolic.

(2) *The relative condition of the vessel (as regards its fullness) during systole and diastole.*—In some cases the systolic portion of the tracing is relatively very much larger than the diastolic. A good example of this condition is seen in aortic regurgitation (see fig. 126), in which the artery is fully distended during systole, but comparatively very empty during diastole.



Pressure $2\frac{1}{2}$ oz.

FIG. 126.—*Aortic Regurgitation.*—Case: G. A., æt. 56, admitted to Newcastle Infirmary 21st February 1878, suffering from shortness of breath and swelling of feet. Had been ill for three months. The face was pale and anxious, lips slightly dusky. Double aortic murmur; heart considerably hypertrophied; apex between 6th and 7th ribs, 3 inches below and 2 inches outside left nipple. Considerable hypertrophy and engorgement of right heart. Died 5th March 1878. Aorta very atheromatous; aortic valves very incompetent; segments shrunken, turned in towards the ventricle; coronary arteries much obstructed; cardiac walls fatty; left ventricle dilated; pericardium adherent. The arteries were practically empty during the ventricular diastole. *a-b*=upstroke; *b*=apex; *c*=tidal wave; *d* indicates the position of the aortic wave, which is absent in this tracing.

An empty condition of the arterial system during the ventricular diastole is often associated with a failing heart, and is a serious indication (see figs. 127 and 128).

When the pulse is *fully dicrotic*, the artery at the point of observation is *apparently*¹ as empty at the end as it is at the beginning of the ventricular systole, and is apparently more empty at the end of the ventricular systole than it is during the ventricular diastole. Again, in hyperdicrotism the artery

¹ See foot note on page 255.

at the point of observation is *apparently* still more empty at the end of the ventricular systole.

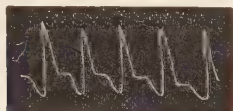


FIG. 127.—Pressure 2 oz.

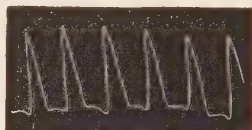


FIG. 128.—Pressure $2\frac{1}{8}$ oz.

FIG. 127.—*Weak Pulse*.—R. R., æt. 17, admitted to Newcastle Infirmary 21st February 1878, suffering from idiopathic anæmia. Died 12th April. There was a systolic (anæmic) mitral murmur. The pulse-tracing resembles that of mitral regurgitation (see fig. 104). Tracing made 23d February.

FIG. 128.—*Progressive Pernicious Anæmia*.—Same patient (see fig. 127). Tracing taken 19th March. The artery is almost empty during diastole.

In other cases on the contrary (in chronic Bright's disease, for example), the diastolic portion of the tracing is unduly sustained. A *persistent* pulse is usually a pulse of high tension, and *vice versa* a pulse which rapidly falls away under the finger is a pulse of low tension.

THE CONDITION OF THE ARTERIAL COATS.

The condition of the arterial coats with regard to the presence or absence of atheroma, is a point of great practical importance, for atheroma of the superficial arteries—the radial, temporal, etc.,—is almost invariably associated with similar disease of the aorta, and very often with disease of the cerebral vessels. The presence or absence of atheroma may be of diagnostic importance, as for example, in a case in which the diagnosis lay between an aneurism and a solid intrathoracic tumour.

Atheroma in the superficial vessels is indicated by:—

(1) Rigidity of the arterial coats; the vessel generally 'stands out like a cord,' is markedly visible and tortuous, while its coats feel thick under the finger. To determine whether the arterial coats are actually thickened or not, the artery should be firmly compressed and emptied by one finger, while the other finger searches for the vessel below the compressed point. When the vessel can be distinctly

felt, even though empty of its contents, its walls are obviously thickened and diseased. This is an important point, for all arteries which are firm and hard, and which stand out like cords, are not thickened.

(2) The presence of a well marked tidal wave, the tension of the pulse being high (*i.e.* the pulse with difficulty obliterated) but a low pressure being usually required for the production of the best tracing (*i.e.* the first ventricular wave being very easily extinguished).

THE COMPARISON OF THE TWO RADIAL PULSES is useful in the diagnosis of some cases of aneurism and intra-thoracic tumour. The two pulses must be compared as regards; (*a*) their synchronism as to time; (*b*) the character of their respective pulse waves.

Differences in time between the two pulses are most easily appreciated by the fingers. In health the two radial pulses are of course synchronous, but in some conditions of disease, as for example, where the sac of an aneurism is situated in the course of the circulation, the pulse wave is retarded, and is consequently delayed at one wrist.

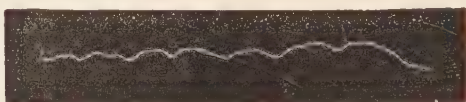
Differences in the character of the pulse waves in the two wrists may also be detected by the finger, but are much more accurately observed by means of the sphygmograph. The precautions which should be taken in comparing the two pulses have been already described. (See page 244.)

Differences in the two pulses may be due to:—

1. Irregular distribution, such as high division of the radial artery. In such cases there is a notable difference in the size of the vessel in the two wrists, or indeed it may apparently be altogether absent on one side. The condition is easily recognised by observing the condition of the brachial arteries on the two sides, and by feeling for the position of the abnormal vessel.

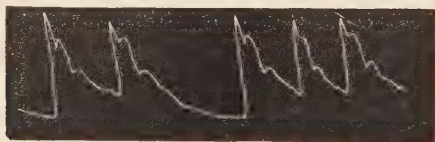
2. The presence of an aneurismal sac in the course of the circulation on one side. The alterations produced by the passage of the blood wave through a globular elastic aneurismal sac, consist in retardation of the pulse-wave and

flattening of its curves. In well marked cases the up-stroke is sloping, the apex rounded, and the secondary curves entirely obliterated. (See figs. 129 and 130, which represent the pulse tracings on the two sides, from a case in which a large aneurism involved the axillary artery on the left side of the body.)



Pressure 3 oz.

FIG. 129.—*Aneurism of Left Axillary Artery (left radial tracing).*—L. G., æt. 63, admitted to the Newcastle Infirmary 7th March 1878, with a large aneurism of the left axillary artery. The apex is rounded; all the curves are obliterated.



Pressure 3 oz.

FIG. 130.—*Aneurism of Left Axillary Artery (right radial).*—Right radial tracing for the same patient. The pulse is intermittent, but all the curves are well marked.

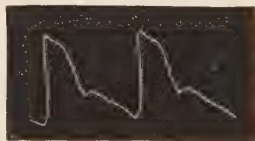
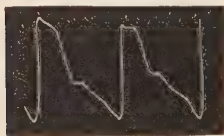


FIG. 131.—(Right radial.) *Pressure $\frac{1}{2}$ oz.* FIG. 132.—(Left radial.) *Pressure $\frac{3}{4}$ oz.*
FIGS. 131 and 132.—*Aneurism of Ascending portion of Aortic Arch.*—J. D., æt. 50, admitted to Newcastle Infirmary suffering from a large aneurism of the ascending thoracic aorta and atheroma. There is no important difference between the two pulses.

Differences in the pulse tracings from the two wrists are not of course observed in all aneurisms. When the aneurism involves the aortic arch below the origin of the innominate the pulse-wave in the two wrists is the same, though the sphygmographic tracing on each side may be modified¹ (each

¹ The alteration in these cases is seldom so great as in aneurisms more peripherally seated (aneurisms of the innominate or subclavian for example).

pulse-wave being affected, *quoad* its curves, in a like degree). Figs. 131 and 132 illustrate this point.

Again, it may so happen that an aneurism is situated on the vessel of each side, or that an aneurism is so filled up with clot that the pulse-wave is very little if at all affected in its passage through it. Such was the fact in a remarkable case of multiple-aneurism which I have recorded in the *Edinburgh Medical Journal* for June 1878, p. 1076. The pulse-tracings from the two radials were in that case almost identically the same. (See figs. 133 and 134.)

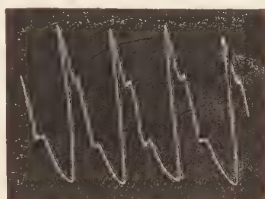
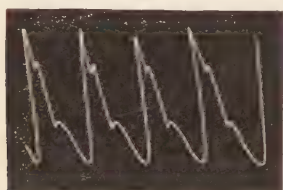


FIG. 133.—(Right radial.) Pressure 3 oz. FIG. 134.—(Left radial.) Pressure 3 oz.

FIGS. 133 and 134.—*Case of Multiple-Aneurism*.—M. F., æt. 64, admitted to the Newcastle Infirmary 18th February 1878, suffering from symptoms of intra-thoracic pressure. Died 10th March. *Post-mortem* showed dilatation and small aneurism of aortic arch. Fusiform aneurisms of the innominate, left common carotid and left subclavian arteries, just above their origins. The aneurisms were filled with firm clots, through which a straight narrow channel for the blood remained.

When the aneurismal sac involves the transverse portion of the aortic arch between the innominate and the left subclavian (the circulation through the innominate being interfered with), the right radial pulse is normal, but the left may be modified.

When the aneurism involves the innominate, the right subclavian, or right axillary artery, the right radial pulse presents the aneurismal characters, while the left is normal. *Vicē versā* when the left subclavian, or left axillary artery is affected, the left radial pulse will be modified, but the right normal.

The alteration in the two radial pulses may be of considerable diagnostic importance. Figs. 135 and 136 represent, for instance, the right and left pulse-tracings of a patient who was admitted to the Newcastle-on-Tyne Infirmary, under

my care, suffering from dyspepsia. There was no complaint of any thoracic trouble. On taking a tracing of the left radial (I was at that time working at the sphygmograph, and taking tracings of every case admitted to hospital) I was, of course, at once struck with its aneurismal character, and on careful physical examination found decided dulness, faint pulsation, and marked accentuation of the cardiac sounds over the chest at a point corresponding to the origin of the left subclavian artery.

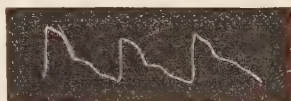


FIG. 135.—(Left radial). Pressure 3 oz. FIG. 136.—(Right radial). Pressure $3\frac{1}{2}$ oz.

FIGS. 135 and 136.—*Aneurism of Left Subclavian.*—J. M., æt. 50, admitted to Newcastle Infirmary 5th September 1878; all the waves in the left tracing are obliterated.

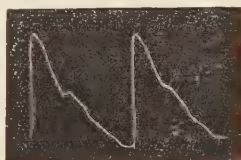
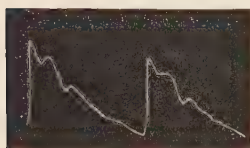


FIG. 137.—(Right radial.) Pressure $2\frac{1}{2}$ oz. FIG. 138.—(Left radial.) Pressure $2\frac{1}{2}$ oz.

FIGS. 137 and 138.—Difference in the radial pulses, the result of pressure by an intra-thoracic tumour on the innominate artery. J. F., æt. 50, admitted to the Newcastle Infirmary 24th January 1878.

3. The pressure of a tumour (solid or aneurismal) on the vessels of one side (on the innominate, or left subclavian for example). (See figs. 137 and 138.)

4. Differences in the calibre of the vessels on the two sides (innominate and left subclavian) such as are produced by obliquity of origin due to disease (aneurismal dilatation of the aortic arch, etc.), or to the presence of an atheromatous patch at the mouth of the vessel or in its course.

5. Local disease in one radial artery.

Asynchronism of the radial pulse with the cardiac contractions has been already alluded to. (See page 268.)

THE EXAMINATION OF THE VENOUS SYSTEM.

I have repeatedly directed attention to the fact that many of the most prominent symptoms in cardiac cases are due to obstructed venous return ; in mitral cases, for example, as soon as compensation fails the whole systemic venous circulation becomes engorged, with the results which will be afterwards described in detail. In cases of this description, the engorgement is, for a time at least, passive ; but should the tricuspid valve become incompetent, either as a secondary consequence of mitral disease or as the result of primary changes in the lungs or right cavities of the heart, a backward or regurgitant current is propelled into the venæ cavæ, and an active obstruction to the venous return is added. Again, in some cases of thoracic aneurism, and in some cases of solid intra-thoracic tumour—conditions which are sometimes with difficulty distinguished—the return current through the superior vena cava is interfered with, and a venous congestion, limited to the territory of that vein, is produced. It is obviously, therefore, of the greatest importance to ascertain, in all cases of cardiac disease, the exact condition of the venous circulation, and this we endeavour to do:—

(1) By observing the colour of the skin of the peripheral parts, more especially of the ears, lips, nose, fingers, toes, etc.—a point which has been considered in treating of the physiognomy of the case (see page 87).

(2) By noting the presence or absence of dropsy in the subcutaneous cellular tissue and serous cavities—a point which has also been considered (see page 89).

(3) By noting the presence or absence of symptoms pointing to venous engorgement of the internal organs, such as the lungs, liver, stomach, kidneys, brain, etc.

(4) By examining the physical condition of the large venous trunks, in particular, the condition of the jugular veins.

Inspection, palpation, and auscultation are the chief means which are employed in order to ascertain the condition

of the superficial veins. When a venous pulse is present valuable information is in some cases afforded by the tracings obtained of it.

Inspection of the veins of the neck.

In conditions of health, more especially in well nourished individuals and during tranquil respiration, the external jugular veins are scarcely, if at all, visible. During forced expiration, more especially in thin persons, they become distended and stand prominently out. All of us have probably noticed the great distention of the veins of the neck in some of our public singers after sustained and powerful efforts.

Distention of the jugular veins is of common occurrence in disease, and is produced by all conditions which interfere with the return of blood to the heart. In practice we find that distention of the jugular veins chiefly depends upon the following conditions :—

1. Mitral lesions.
2. Diseases of the lungs (such as cirrhosis, emphysema, etc.) which interfere with the passage of the blood from the right to the left sides of the heart.
3. Disease of the right side of the heart.
4. The pressure of aneurisms and solid intra-thoracic growths upon the superior vena cava.
5. Pericarditis and inflammation of the cellular tissue of the mediastinum.—In some cases of adherent pericardium and of indicative mediastino-pericarditis, the current of blood to the heart is interfered with and the jugular veins are distended.
6. Plugging of the superior vena cava—an extremely rare condition, which is generally due to disease of the venous coats, produced by external pressure, *e.g.* the pressure of an aneurism or intra-thoracic growth.

In distention of the jugulars due to the central causes, *i.e.* to disease of the heart or lungs, the distention is not constant but varies with the condition of the respiration, being most marked during expiration, least marked and usually com-

pletely absent during inspiration. (The distention of the veins of the neck, which is due to adherent pericardium, occurs during the depression of the chest wall which corresponds to the systole of the ventricles; the distention of the veins rapidly collapses and disappears during the diastole of the ventricles. The distention is due to the fact, that during the systolic depression of the chest wall, the cavity of the chest is diminished in size, and a mechanical hindrance offered to the return current of blood through the superior vena cava. During the diastolic rebound, on the other hand, the cavity of the chest is suddenly distended, a suction force is exerted, and the previously engorged veins are, in consequence, suddenly emptied. In indurative mediastino-pericarditis, the distention of the veins of the neck is due to the pressure of bands of inflammatory adhesions upon the vena cava superior; and since the obstruction only occurs during inspiration, the jugular veins become distended, instead of collapsing, during inspiration, as they do in health; the distention rapidly subsides during expiration, that is, when the constrictive bands are relaxed, and the obstruction to the blood in the superior cava is removed.) Its amount, too, varies with the position of the patient. When sitting or standing, the distention is less than when lying down, owing to the fact that in the erect position of the head, the return of the venous blood to the heart is favoured by the force of gravity. The distention is usually greater on the right, than on the left side of the neck, and is generally much more apparent in the external than in the internal jugulars. Exceptions to both of these statements do, however, occasionally occur.¹

The swollen external jugular veins have often a knotted appearance, the irregular enlargements corresponding to the position of the venous valves.

As the result of distention of the external jugulars, the face (more especially the lips, ears, and nose) becomes cyanotic, and the features swollen, but the swelling is never very

¹ I have seen a case of tricuspid regurgitation in which the distention and pulsation in the internal jugulars were much greater than in the external.

great.¹ In consequence of the venous distention, the brain becomes congested, and symptoms of carbonic acid poisoning, viz., drowsiness, mental obfuscation, and ultimately coma, are apt to supervene.

When the distention is due to a central cause, *i.e.* to disease of the heart or lungs, the venous engorgement is not limited to the territory of the superior cava, but its effects are also seen in the lower extremities and great organs of the abdomen, *i.e.* in the territory of the inferior cava. Indeed, as we have previously seen, swelling of the feet is the first symptom in many of these cases, and is generally observed before the distention of the jugulars is noticeable.

When the distention depends upon a local, as distinguished from a central cause, *i.e.* upon direct obstruction to the return current through the superior cava, the symptoms and signs of venous engorgement are of course limited to the two sides of the head and neck, the upper extremities, and the upper parts of the thoracic parieties. When the obstruction is great, and particularly in those cases in which it is complete, the parts from which the superior cava draws its blood may become remarkably œdematous, and it is in cases of this description that the peculiar, hard, brawny swelling, to which the term 'collar of flesh,' has been applied, is observed.

As I have previously pointed out, the swelling of the face, which is due to obstructed venous return through the superior cava is readily distinguished from that which is met with in renal dropsy, and with which it might at the first glance be confounded, by the facts :—

- (1) That the face is not only swollen but is also cyanotic.
- (2) That the swelling involves, and is limited to, the territory of the superior cava.
- (3) That there is no kidney disease.
- (4) That there are symptoms and signs of aneurism, intra-thoracic growth, or other local pathological condition likely to obstruct the vein or produce disease of its coats.

¹ The swelling is never so great as in cases of obstruction of the superior vena cava from direct pressure.

Pulsation in the jugular veins.—In conditions of health there is no perceptible pulsation in the jugular veins, but in cases of disease, rhythmical undulations and pulsations, more particularly in the external jugulars are sometimes met with. In some cases these pulsations are only apparent; in others they are real, and represent a true venous pulse.

Apparent pulsation.—When the jugulars are distended, as the result of a central obstruction, *i.e.* of disease of the heart or lungs, a rhythmical undulation, corresponding in time to the movements of inspiration and expiration, is generally observed. The vein is distended during expiration and collapses during inspiration. This apparent pulsation is at once distinguished from a true venous pulse by its rhythm.

In indurative mediastino-pericarditis, as has been described above, an 'apparent' pulsation is sometimes seen in the veins of the neck; but it differs from the ordinary form of apparent pulsation, inasmuch as the vein is distended during inspiration and empty during expiration.

In some cases of adherent pericardium, the veins of the neck are distended during the systole of the ventricles, as I have described above. This pulsation, which is of course only apparent and due to simple passive distention, must be distinguished from true pulsation of tricuspid regurgitation in which a 'back-wash' is propelled into the veins at each systole of the right ventricle.

In other cases, undulations which correspond in time to the cardiac contractions are observed. In some of these the undulations are due to impulses communicated to the distended veins by the subjacent arteries; in others, they are due to the fact that the contraction of the right auricle communicates an impulse or shock to the blood in the superior vena cava, which is propagated to the jugulars. It is also said that the contraction of the right ventricle, even when the tricuspid is sound, may communicate a shock through the tightly stretched tricuspid valve, to the blood in the right auricle, which may be propagated, through the vena cava superior, to the jugulars. It is improbable, however, that any marked undulation in the jugulars can be produced in this way.

Apparent pulsation of this description, *i.e.* apparent pulsation of the jugular veins synchronous with the contraction of the heart, is readily distinguished from true jugular pulsation produced by a back-wash, *i.e.* from pulsation indicative of tricuspid regurgitation, by compressing the vein in the middle of the neck, and observing the condition of the portion of vein which is placed below the finger, *i.e.* between the heart and the compressed point. If the pulsation is real, it will still be observed in the vein below the finger, for with each systole of the right ventricle the vein is again filled from below by the back-wash. If the vein remains collapsed and empty, the pulsation is only apparent.

True pulsation of systolic rhythm is due to a stream of blood being propelled into the vein at each systole of the right ventricle. This condition is of extreme importance, and indicates tricuspid incompetence; it also shows that the valves at the point of junction of the internal jugular and subclavian veins are incompetent. Jugular pulsation of this description (*i.e.* true jugular pulsation indicative of tricuspid regurgitation), is sometimes associated with pulsation of the inferior vena cava, the effects of which have been previously described.

The exact character of the jugular pulse-wave is, as Friedreich has shown, of some practical importance. In an extremely able paper on 'The Auricle in Health and Disease,' Dr Gibson has directed attention to this point; and has figured some of Friedreich's tracings (see figs. 139, 140) 'The

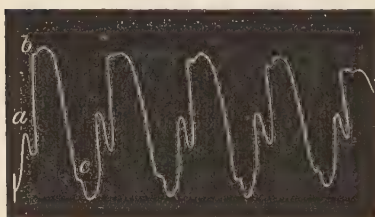


FIG. 139.

FIG. 139.—Jugular pulse tracing showing the presence of the auricular wave *a*. (*After Friedreich.*)

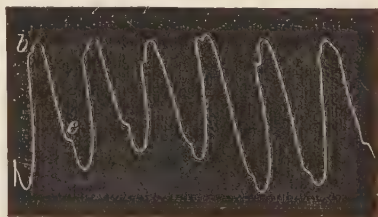


FIG. 140.

FIG. 140.—Jugular pulse tracing after paralysis of the auricle; the auricular wave *a*, in the preceding figure is no longer present. (*After Friedreich.*)

one shows,' he says, 'three waves, *a*, *b*, and *c*; the other only two, *b* and *c*. Bestowing our attention mainly upon the first wave, *a*, which is caused by the systole of the right auricle, we find in it an index of the state of the auricular muscle. So long as it is shown in tracings from the jugular, the auricle is comparatively healthy; when it disappears it is a certain proof of paralysis of the auricle, and if it should reappear it gives evidence of a return of contractile power. In this way the auricular wave in the jugular veins is of very considerable practical interest. The large wave, *b*, is caused by the systole of the ventricle, and the third wave, *c*, is reflected from the interior of the heart, in the same way as the dicrotic wave in the radial tracings of aortic regurgitation first shown by Naumann.'¹

True pulsation of presystolic rhythm.—When the venous valves are incompetent in consequence of over-distention of the veins, pulsation of presystolic rhythm is sometimes observed. It is produced by the contraction of the right auricle and may occur independently of any systolic 'backwash' representing tricuspid regurgitation.

Pulsation, like distention, is usually more marked in the right than in the left side of the neck, and is generally more perceptible in the external than in the internal jugulars.

Pulsation in the peripheral veins, such as the veins on the back of the hand, is occasionally though rarely observed. It is seen, for instance, in some cases of aortic regurgitation, and is due to the fact that the pulse wave is not obliterated in the arteries as in health, but passes right on to the veins through the capillaries. Prof. Michael Foster gives the following lucid description of the ways in which it may be produced:—'To recapitulate: there are three chief factors in the mechanics of the circulation, (1) the force and frequency of the heart-beat, (2) the peripheral resistance, (3) the elasticity of the arterial walls. These three factors, in order to produce a normal circulation, must be in a certain relation to each other. A disturbance of these relations brings about abnormal conditions.

¹ *Edinburgh Medical Journal*, Aug. 1882, p. 126.

Thus, if the peripheral resistance be reduced beyond certain limits, while the force and frequency of the heart remain the same, so much blood passes through the capillaries at each stroke of the heart that there is not sufficient left behind to distend the arteries, and bring their elasticity into play. In this case the intermittence of the arterial flow is continued on into the veins. An instance of this is seen in the experiments on the sub-maxillary gland, where sometimes the resistance offered by the minute arteries of the gland is so much lowered, that the pulse is carried right through the capillaries, and the blood in the veins of the gland pulsates. A like result occurs when, the peripheral resistance remaining the same, the frequency of the heart's beat is lowered. Thus the beats may be so infrequent that the whole quantity sent on by a stroke has time to escape before the next stroke comes. Lastly, if, while the heart's beat and the peripheral resistance remain the same, the arterial walls become more rigid, the arteries will be unable to expand sufficiently to retain the surplus of each stroke or to exert sufficient elastic reaction to carry forward the stream between the strokes; and in consequence more or less intermittence will become manifest.¹

Palpation of the superficial veins is not a method of physical examination of much consequence, except in those cases in which the vein is plugged by a coagulum. In cases of this description, the vein can be felt as a firm rigid cord, and there is usually at the same time tenderness on pressure. In cases of well marked venous pulsation, such for instance as is due to tricuspid incompetence, the pulsations can be felt as well as seen. In some cases of anæmia, in which the venous hum which I shall presently describe is well marked, a thrill can be felt when the finger is placed over the veins at the root of the neck.

Auscultation of the veins.—In a considerable proportion of cases, even in apparent health, and notably in conditions of anæmia, a continuous humming murmur, to which the term

¹ *A Text Book of Physiology.* Fourth edition, p. 131.

bruit de diable, or *humming-top murmur*, has been given, is heard in the veins. In many cases it closely resembles the roaring sound which is heard when a shell is placed close to the ear. It is usually best heard at the junction of the internal jugular and sub-clavian veins, is intensified by the pressure of the stethoscope, by turning the head forcibly to the opposite side,¹ and by a deep inspiration.²

The murmur is generally louder on the right side. It is usually continuous, but in some cases is only produced on deep inspiration, and in such cases is, therefore, intermittent.

In conditions of anæmia venous murmurs may be often heard in other situations, as, for example, over the torcular Herophili and eyeball.

¹ When the head is turned to the opposite side, the muscles and fascia are put upon the stretch, the vein is compressed, and its channel narrowed.

² A deep inspiration quickens the flow of blood to the heart, and favours the production of fluid veins.

CHAPTER IV.

DISEASES OF THE PERICARDIUM; ACUTE PERICARDITIS; PERICARDIAL ADHESION; INDURATIVE MEDIASTINO-PERICARDITIS; CHRONIC PERICARDITIS; HYDROPERICARDIUM; PNEUMOPERICARDIUM.

HAVING made ourselves acquainted with the introductory parts of our subject (the anatomy, physiology, pathology, and clinical examination of the heart), we are now in a position to study the individual diseases in detail. It will be convenient to consider:—

- (1.) The diseases of the pericardium.
- (2.) The diseases of the endocardium.
- (3.) The diseases of the myocardium.
- (4.) The neurotic affections of the heart, which include the purely functional disorders of the organ.
- (5.) The diseases of the great blood-vessels.

The congenital malformations of the heart, which are usually treated under a distinct head, will be described under the diseases of the endocardium and myocardium.

THE DISEASES OF THE PERICARDIUM.

The morbid conditions of the pericardium, which are of practical and clinical importance are:—

1. The inflammatory affections and their results (pericarditis, adherent pericardium, indurative mediastino-pericarditis).
2. The effusions of fluid (of a non-inflammatory character) into the sac (hydropericardium, hæmopericardium).

3. The collections of air or gas in the sac (pneumopericardium).

Congenital deficiencies and malformations (diverticula) of the pericardium occasionally occur, but are seldom suspected or recognised during life. In a few cases the whole pericardial sac has been absent, more frequently, limited deficiencies or imperfections in it have been observed.

New growths (tubercles, sarcomatous and cancerous tumours) occasionally affect the membrane; and parasites and free bodies (which usually have resulted from previous inflammatory deposits or new growths) are very exceptionally found in the sac. But it is not necessary to describe these conditions separately. Unless they give rise to pericarditis (which they frequently do) they are unattended by any distinct symptoms or physical signs, and cannot be recognised during life.

PERICARDITIS.

Definition.—Inflammation of the pericardium.

Like all other forms of inflammation, pericarditis may be either acute, sub-acute, or chronic. As a matter of practical convenience it is only necessary to describe separately the acute and chronic forms.

ACUTE PERICARDITIS.

Definition.—Acute inflammation of the pericardium.

Varieties.—Many different varieties of the disease may be described. We may, for example, make an ætiological subdivision, and divide cases of pericarditis into two great groups, viz. *primary* and *secondary*, and then again subdivide each of these groups into still smaller ones, as, for instance, into cases of *traumatic* pericarditis, *rheumatic* pericarditis, *idiopathic* pericarditis, and the like. Or, we may make a pathological division, in accordance with the extent of the inflammation (*diffuse* and *circumscribed*¹ pericarditis), or in accordance with the character of the inflammatory product (*fibrinous*, *purulent*,

¹ The division into diffuse and circumscribed is theoretical rather than practical, for in acute pericarditis, at all events, the inflammation is almost invariably diffuse.

hæmorrhagic, etc). Or again, we may take a clinical (or rather a clinico-pathological) basis, and divide cases of pericarditis into *dry* and *moist*, in accordance with the amount of effusion and the condition of the cardiac dulness.

Ætiology.—Acute pericarditis may occur at any period of life, but it is rarely met with in young children; it is most common between the ages of fifteen and twenty-five. It is slightly more common in men than in women, and is most frequently observed in persons who follow outdoor occupations, more especially those who are exposed to cold and wet, and to great variations in temperature; facts which are probably explained by the circumstance that men, and especially men who are exposed to cold and wet, are more frequently affected with acute rheumatism than other members of the community. In women it is much more common amongst young servant girls and washer-women than in those engaged in any other occupation. Over-action of the heart seems to predispose to the condition.

Primary pericarditis is extremely rare. It may be due to:—

(a) Traumatic injuries, such as wounds of the sac, blows on the front of the chest, etc.

(b) Primary new growths (sarcomata, carcinomata, etc.) in the pericardial tissues. These primary new growths are extremely rare.

(c) Exposure to cold and wet, independently of acute rheumatism or of any other diseased condition. This is probably the rarest of all the causes of pericarditis. The following is a case in point; it is also interesting from the fact that until shortly before death there were no symptoms indicative of serious disease.

Case—Latent idiopathic pericarditis; death.

A girl, æt. 17, who had previously enjoyed good health, but who had never menstruated, complained of uneasiness in the upper part of the abdomen, and of constipation. The tongue was slightly furred, the pulse

a little quick, but there was nothing to direct attention to any serious disease within the chest. A dose of castor oil was prescribed, and nothing more was heard of the case for a few days. I was hurriedly sent for, and found the patient dead. She had passed a somewhat restless night, but had got up to breakfast as usual; had suddenly complained of feeling faint, and had fallen back dead. All the organs were healthy, with the exception of the heart. The pericardium was distended with turbid fluid, and both surfaces of the pericardium—both visceral and parietal layers—were covered with a layer of recent lymph. (See fig. 141.) The case was therefore one of pericarditis; and syncope was the immediate cause of death. On inquiry I ascertained that the patient had waded in the sea a fortnight previously, and that for some days afterwards she had been a little hoarse. There was no history of rheumatism. So trivial were the symptoms, in this severe and acute case, that the patient was able to go about her occupation—that of a baker's assistant—until two days before her death.

Secondary pericarditis is a comparatively common condition. It occurs:—

(a) In the course of some general affections. The disease *par excellence* with which it is associated, and of which it is, as it were, part and parcel, is acute rheumatism. Indeed, a large proportion of the cases of acute pericarditis, met with in practice, are rheumatic. It is more apt to occur in first than in subsequent attacks of rheumatic fever, and in severe rather than in mild cases. Pericarditis also occasionally occurs in the course of scarlet fever. It is a somewhat common complication in Bright's disease, more especially the cirrhotic form; and in cases of that description is not unfrequently the immediate cause of death. It is sometimes met with in pyæmia, purpura, and scurvy.

(b) As the result of the direct extension to the pericardium of an inflammation or new growth which has originated in some of the adjacent organs or parts. This is one of the most common, probably *the* most common cause of pericarditis which proves fatal during the acute stage.¹ The primary inflammation or new growth may originate in the pleura, lung, bronchial glands, tissues of the mediastinum, the cellular tissue of the neck, or in the structures below the

¹ Rheumatic pericarditis is, perhaps, relatively more frequent, but is very seldom fatal.

diaphragm, *i.e.* in the cavity of the abdomen. An abscess of the liver, for example, may by direct extension through the diaphragm (and without bursting into the sac) give rise to an inflammation of the pericardium.

(c) As the result of secondary deposits of a tubercular, sarcomatous, or cancerous nature in the structure of the pericardium, the primary new growth being situated at a distance, and the secondary deposits being carried to the pericardium by the veins or lymphatic vessels.

Pathology and Morbid Anatomy.—The naked eye and microscopical appearances which are seen in cases of acute pericarditis vary with :—

- (1.) The extent of the inflammation.
- (2.) The stage of the inflammation.
- (3.) The amount and kind of exudation.

The extent of the inflammation.—In the great majority of cases the inflammation is general, and involves the whole extent of both layers of the pericardial sac. In rare cases the inflammatory process is limited in distribution. In recent cases of this description, the chief morbid appearances are usually to be found at the base of the organ, around the roots of the great vessels and auricular appendages. In other cases, more particularly when the inflammation is of some duration or has become chronic,¹ inflammatory adhesions, of limited extent, are found at or near the apex, on the posterior surface, or it may be on any part of the organ.

The stage of the inflammation, and the amount and kind of exudation.—In pericarditis the primary vascular changes (dilatation and stasis) which characterise all inflammations are quickly followed by the pouring out of inflammatory exudation matters into the serous cavity or sac.

The naked eye appearances are as follows :—

In the *earliest stages*, which it must be observed we seldom

¹ To avoid repetition, the pathological appearances which are seen in sub-acute and chronic pericarditis are included under this description.



FIG. 141.

Heart from a case of recent Pericarditis. (Natural size.)

The surface of the organ is coated with lymph, which is easily detached. The letter, a, points to a portion of the heart covered by lymph; b, to a detached shred of lymph; c, to a portion of the heart from which the lymph has been removed.



FIG. 142.—*Front view of the heart in a case of acute pericarditis. (Natural size.)*

The front of the parietal pericardium, which was very much thickened, has been cut away; a wedge-shaped portion has been removed from the apex of the left ventricle for microscopical examination.

Note.—To be seen properly this drawing should be held about two feet from the eye.



FIG. 143.—Heart in a case of acute pericarditis; its surface is covered with long fibres of shaggy lymph, giving it a hairy appearance; the parietal portion of the pericardium has been turned upwards. (Natural size.)

Note.—To be seen properly this drawing should be held about two feet from the eye.

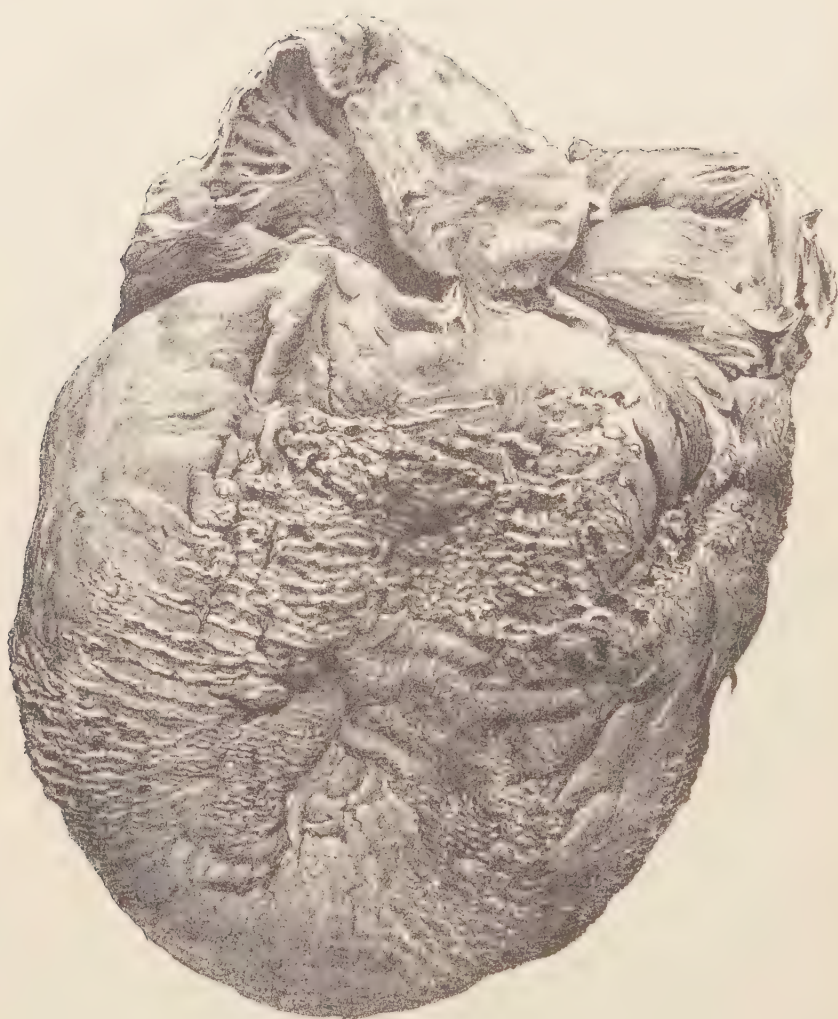


FIG. 144.

Heart from a case of Pericarditis, showing vegetations consisting of firm lymph on its exterior.
(Natural size.)

have an opportunity of studying on the *post-mortem* table, the membrane is seen to have lost its normal smooth, glistening, appearance; its vascularity is, in places at least, increased; and small punctiform ecchymoses are sometimes to be seen. In the great majority of cases small deposits of fibrine can generally be perceived by the naked eye; and the normally smooth membrane has a more or less rough feel when the finger is passed over it.

In a few hours, the exudation becomes more copious. The appearances, which are seen when the *full development* of the exudation is reached, vary greatly in different cases.

In some cases the amount of fluid poured out into the pericardial sac is scanty, in others copious. In most cases the exudation is sero-fibrinous, and instead of the clear serum which is normally found in the sac of the pericardium after death, the fluid is more or less turbid, and contains flakes of coagulated lymph. In others it is blood tinged, much more rarely it is bloody or purulent.

Both layers of the pericardium are now generally coated with a thick layer of lymph; and punctiform ecchymoses, or in some cases larger extravasations of blood, are to be seen. In consequence of the movements of the heart (*i.e.* of the opposed surfaces of the pericardium, which are coated with exudation matters), the exudation always presents a rough, ragged, or papillated appearance. (See figs. 141, 142, 143, 144.) In some cases, it is thrown into ridges, in others it is honey-combed, and in many it closely resembles the appearance which is produced by forcibly separating a thickly spread sandwich of bread and butter; in others, the lymph is drawn out into fine threads or strings, and the exterior of the heart presents a shaggy appearance—the so-called *hairy* heart. (See fig. 143.)

In the earlier stages of acute cases the exudation materials can be readily detached from the surface of the membrane, leaving a smooth surface underneath. (See fig. 141.)

In some cases cancerous or tubercular deposits (usually miliary tubercles) are seen on the surface of the membrane. In these cases the exudation is often hæmorrhagic. In

purpura and scurvy, and in other cachectic conditions, the exudation also tends to be bloody.

In purulent pericarditis the lymph is less gelatinous, more creamy-looking and opaque. The exudation is generally purulent from the first; occasionally a sero-fibrinous exudation becomes purulent. Cachectic conditions, pyæmia, puerperal fever, the bursting of an abscess into the pericardium, etc., are the conditions with which a purulent inflammation of the pericardium is usually associated.

After the exudation has lasted a longer or shorter period, absorption and organisation occur. The fluid parts of the exudation disappear, the two surfaces of the pericardium come in contact, and adhesions are usually developed. In some cases the whole pericardial sac is obliterated, frequently the adhesions are partial, occasionally they are limited to small areas—such as the roots of the great vessels or the apex. In quite exceptional cases an example of which has recently come under my observation, the inflammatory exudation is absorbed without the formation of adhesions. In cases of this description, limited opacities and thickenings of the membrane which it is difficult, or impossible to distinguish from the so-called ‘milk-spots,’ which are so frequently met with, may be seen.

These so-called ‘milk-spots’ are most frequent over the anterior surface of the right ventricle, at the apex, or at some other part of the organ which is exposed to friction and pressure. They are much more common, therefore, in large dilated or hypertrophied hearts (*i.e.* in those cases in which the lungs acting as it were as buffers or water cushions, are pushed aside, and the enlarged heart comes in contact with the chest wall). They consist of localised thickenings of the superficial layers of the pericardium. (See fig. 234.) They are undoubtedly due to the long continued irritation which is produced by intermittent pressure.

Very exceptionally calcareous salts are deposited when the exudation matters are absorbed; and in a few cases, a large portion of the heart has been surrounded with a dense calcareous covering. This result is more likely to occur after

purulent than after simple (*i.e.* non-purulent) pericarditis. Calcareous plates and deposits would probably be more frequent if it were not for the facts, *firstly*, that purulent pericarditis is rare, and, *secondly*, that the great majority of cases terminate fatally during the stage of suppuration, *i.e.* before calcareous changes in the exudation matters can occur.

The microscopical appearances of pericarditis.—Before describing the appearances which are seen under the microscope in pericarditis it will perhaps be well to direct attention to the normal histology of the membrane.

A section through the visceral layer of the pericardium and of the cardiac wall which is situated beneath it (see fig. 145) is composed of the following parts:—

(1.) *The epithelial layer*, which consists of a single layer of pavement epithelial cells.

(2.) *The fibrous layer*, which is composed of fibrous tissue.

Immediately beneath the epithelium the fibres, which are ordinary connective tissue fibres, are arranged parallel to the surface; in the deeper parts of the fibrous layer they form a net-work, and in this part of the membrane elastic fibres are found.

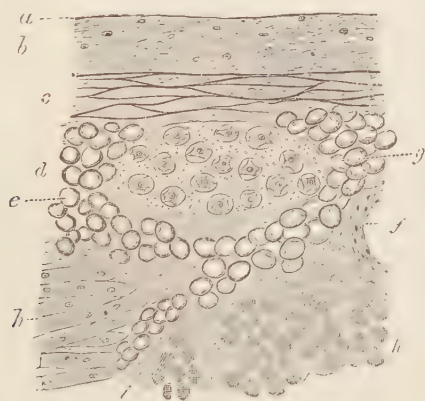


FIG. 145.—Section of a part of the pericardium of the right auricle. (After Quain.)

a, serous epithelium in section; *b*, connective tissue layer; *c*, elastic network; *d*, sub-serous areolar tissue; *e*, fat; *f*, section of a blood-vessel; *g*, a small ganglion; *h*, muscular fibres of the myocardium; *i*, inter-muscular areolar tissue.

(3.) *The sub-serous layer*, which consists of areolar tissue and fat cells, and in which the blood-vessels and nerves (ganglia and nerve trunks) are situated.

The sub-serous connective tissue is continuous with the connective tissue which passes between the muscular fibres in the cardiac wall—a connection of some importance, for it explains the facility with which inflammation travels from the pericardium to the myocardium, and even to the endocardium.

The amount of fat varies considerably in different cases; in spare individuals it is generally limited to the furrows which contain the larger blood-vessels, but in fat persons it may form a layer—sometimes of considerable thickness—over the whole, more particularly over the anterior surface, of the organ.

The ganglia are most numerous in the neighbourhood of the large vessels, more especially in the sulci which separate the auricles from the ventricles.

Now in the *earliest stages* of pericarditis the following changes are found:—

(1.) The blood-vessels are dilated and engorged with blood.

(2.) An exudation of fibrinous lymph is seen on the surface of the epithelial layer, in the deeper parts of the fibrous layer, and in the sub-serous areolar tissue. The fibrinous lymph on the surface of the membrane is, even in the earliest stages in which we have an opportunity of examining it, arranged in the form of irregular projections of various shapes and sizes. (See figs. 146 and 147.) These projections consist of amorphous or finely granular fibrine in which leucocytes and a few larger cellular elements, derived from the epithelium, can generally be distinguished. The inflammatory exudation is very loosely attached to the surface of the pericardium, and is readily detached in the process of preparation.

(3.) Detachment and proliferation of the epithelium. This change, which is well seen in fig. 148, is sometimes difficult to make out in sections, but cells of various shapes and sizes, which are undoubtedly derived from the epithelium, can



FIG. 146.—Section through the wall of the left ventricle in early pericarditis showing small papillæ of lymph on the surface of the pericardium. (Magnified about 10 diameters.)

e, endocardium; *m*, myocardium; *p*, *p*, pericardium; *l*, *l'*, lymph.



FIG. 147.—A portion of the same preparation more highly magnified (about 50 diameters).

m, muscular fibres of the myocardium; *p*, pericardium; *a*, the papilla of lymph to which the letter *l'* in figure 146 points.

Note.—To be seen properly this figure should be held about four inches from the eye.

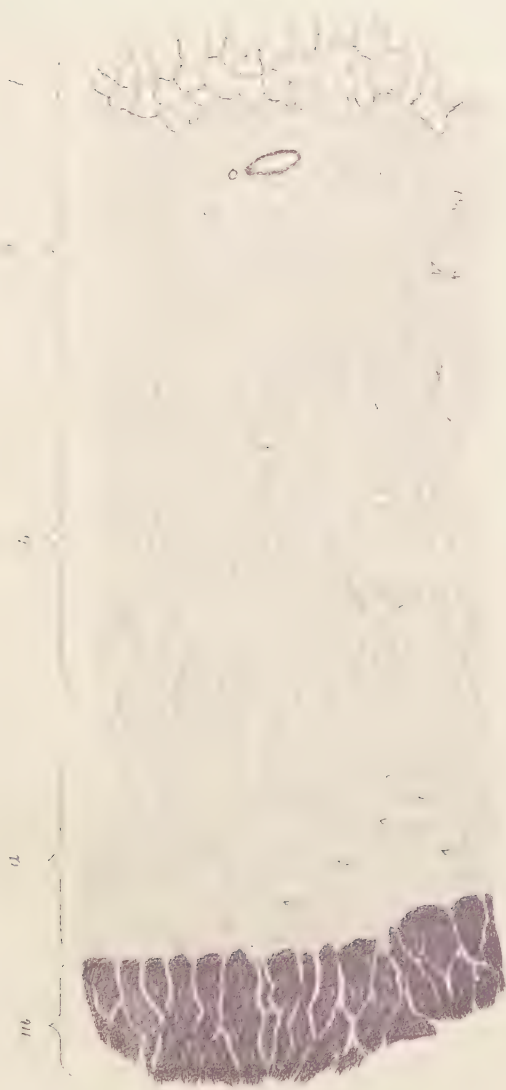


FIG. 149.—Section through an adherent pericardium, magnified about 10 diameters.

m, muscular fibres of the heart; *a* and *a'*, layers of organising tissue; *b*, lymph undergoing absorption.

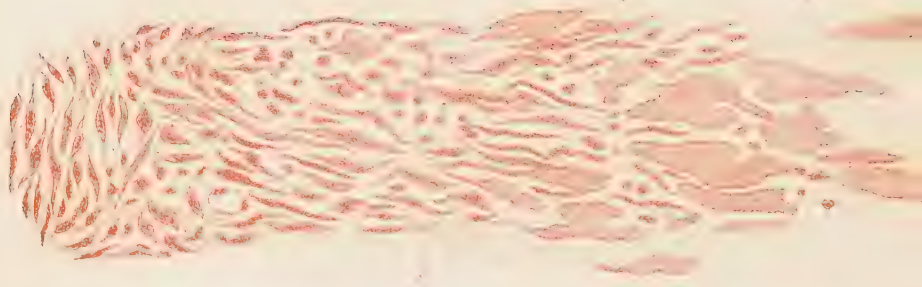


FIG. 150.—A portion of the preparation shown in fig. 149 more highly magnified, about 250 diameters, showing the junction of the spindle celled tissue, and the lymph layer. Large masses of lymph (*l, l*) are becoming converted into spindle cells (*s, s*.)

FIG. 151.

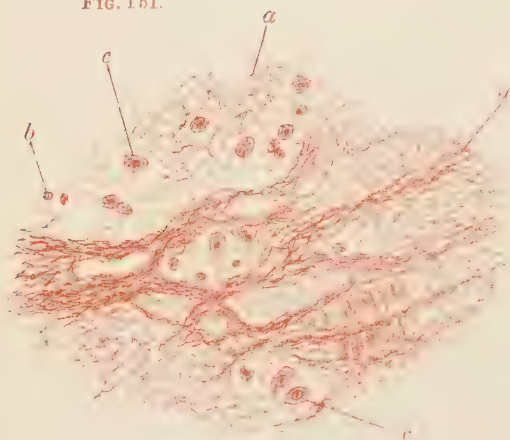


FIG. 151.—A portion of the central lymph layer of the preparation shown in fig. 149, magnified about 200 diameters, showing lymph threads (*a*), leucocytes (*b*), and large epithelial cells (*c*).

FIG. 152.



FIG. 152.—Another portion of the central lymph layer of the preparation shown in fig. 149; showing collections of red blood corpuscles (*a*), surrounded by lymph threads (*b*).

usually be seen in the meshes of the exudation, and in the fluid contents of the pericardial sac.

(4.) An exudation of leucocytes, and in some cases of red blood corpuscles in the lymph on the free surface of the membrane, and in the deeper parts of the fibrous and sub-serous layers.

At the height of the exudation the layer of fibrinous lymph on the surface of the pericardium is much increased in thickness, and the fibrous and sub-serous layers of the pericardium are more swollen, the exudation corpuscles being proportionately more numerous. After a little time new blood vessels are formed, often in great abundance, in the thickened fibrous layer; and the rupture of these delicate vessels may be the source of the hæmorrhagic extravasations which are so frequently seen in the inflamed membrane.

In a considerable proportion of cases the myocardium is implicated. The muscular fibres, which are situated immediately beneath the sub-serous layer, are very generally affected—and this is more especially the case where the inflammation is severe and has lasted for any time. The muscular fibres are swollen; their transverse striæ are indistinct; they stain badly with picro-carminé; and in some cases they are distinctly fatty. Fibrinous lymph, leucocytes, and (in many cases) red blood corpuscles are seen amongst the areolar fibres and around the fat cells, which pass from the deeper surface of the pericardium into the myocardium, and in the spaces which separate the individual fibres.¹

In the stage of absorption and resolution the fluid parts of the exudation are removed by absorption, the exudation products undergo fatty degeneration, and numerous fine oil globules are seen in the affected part; many of the corpuscular elements in the thickened fibrous and sub-serous layers are developed into spindle cells, and ultimately into fibrous tissue. Blood vessels shoot out from the surface of the membrane into the substance of the exudation; and adhesions, composed of spindle cells and fibres, containing

¹ The fatty changes in the muscular fibres are not generally observed unless the inflammation has lasted some little time.

blood vessels, and in some cases covered by a beautiful layer of epithelium, are formed between the two opposed surfaces of the sac. In this way the pericardium may be ultimately divided into a large number of secondary sacs all lined with epithelium.

In those cases in which the adhesions are universal, the sac of the pericardium is completely obliterated. A thick, dense, fibrous layer, consisting of spindle cells, fibres, and containing numerous blood vessels, is in many of these cases, found on the surface of the heart. The microscopical appearances which pericardial adhesions present, are shown in figs. 149 to 157.

When the myocardium is at the same time implicated, fibrous tissue may be formed in considerable quantity between the muscular fibres, which undergo fatty degeneration and atrophy; but these changes, which are of the greatest practical importance from a clinical point of view, and the secondary changes in the heart which result from adhesions of the pericardium, will be more appropriately considered afterwards.

The fibrous layer of the parietal pericardium is much less frequently implicated in pericarditis than the fibrous layer of the portion of the membrane which covers the heart; but in some cases the inflammation not only involves the whole thickness of the parietal pericardium, but also extends to the adjacent structures—the pleura, the cellular tissue of the mediastinum, or even the lung itself.

The Clinical History of Acute Pericarditis.

In describing the clinical history of acute pericarditis, it will be convenient to consider, in the first place, the individual symptoms and physical signs, and then to give a brief general description of the chief types of the affection.

Symptoms.—In order to understand the symptoms of acute pericarditis, it is important to remember that, in a large proportion of cases the pericardial inflammation is secondary; and that, in cases of this description, the symptoms of the primary disease are often very striking—so prominent, indeed, as to obscure those which result from the inflammation of the pericardium itself.



FIG. 153.—A portion of the spindle-celled layer from the section represented in fig. 149, more highly magnified (about 250 diameters).

a, spindle cell; *b*, capillary vessel; *c*, leucocyte.

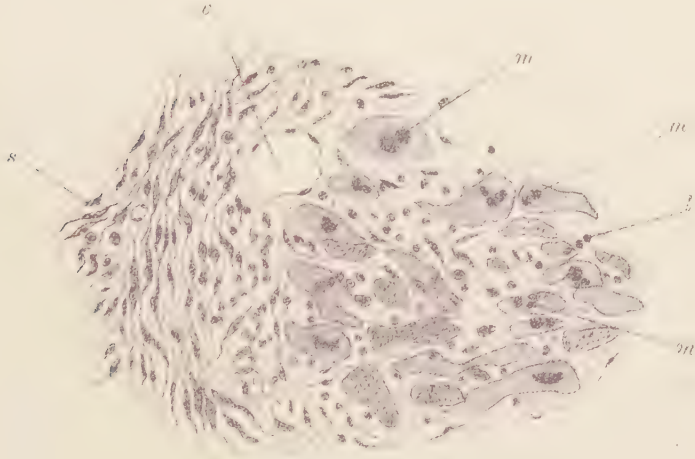


FIG. 154.—A portion of the preparation shown in fig. 149, at the junction of the myocardium and spindle-celled layer, magnified about 200 diameters; showing leucocytes between the muscular fibres, proliferation of the nuclei of the muscular fibres and disappearance of their fibrillae.

s, spindle cells; *v*, vessel transversely divided; *m*, *m*, proliferating nuclei of the muscular fibres.



On 12 May 1994, the first of a series of 12 days of cold and dry, unimpaired weather began.

where a is a part of the various permeability, b, b' denote connective tissue bundles passing through the pericardial space, c, c' are the visceral pericardium which is not shown in the diagram, d, d' are bundles of connective tissue, e, e' collections of epineurial cells, f, f' blood vessels.

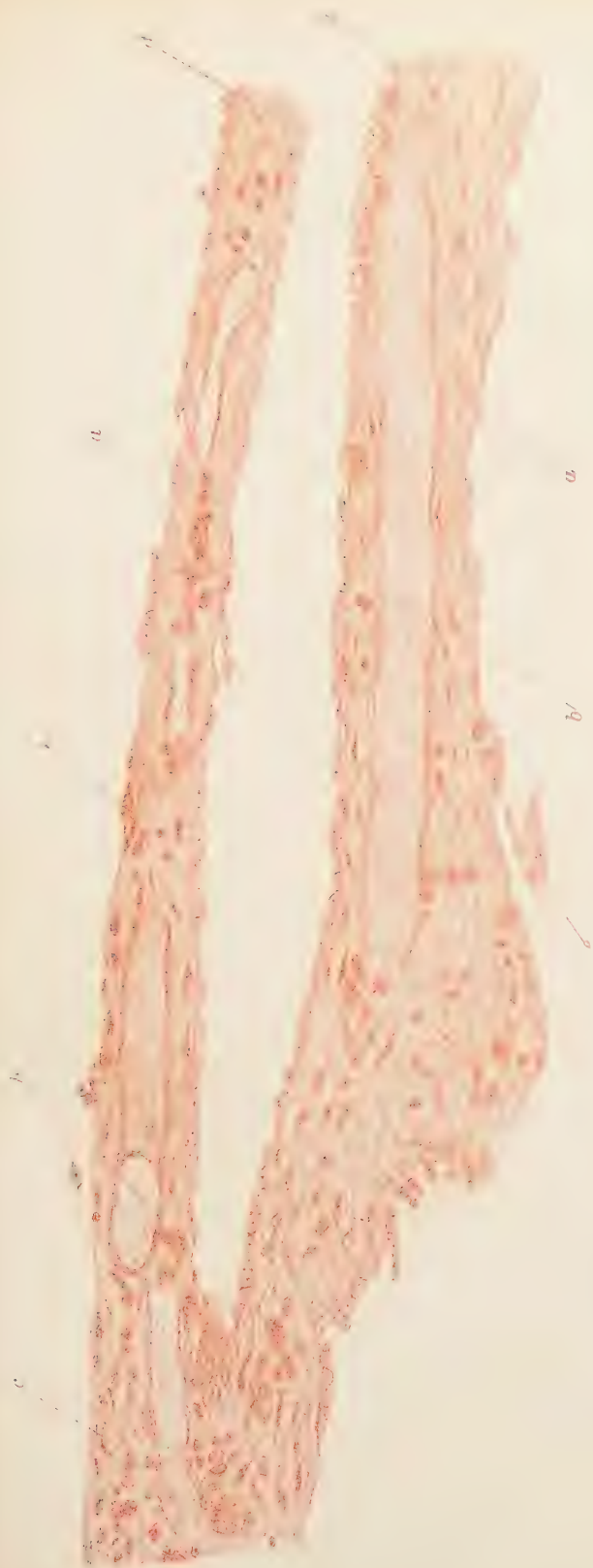


FIG. 10. Longitudinal section of a blood vessel, showing the process of adhesion. The adhesion is covered with a layer of cells of epithelial and connective tissue. *a*, the adhesion, which has become detached; *c*, the cardio, or the vessel, at the point of adhesion shown in the drawing; *b*, blood-vessels.

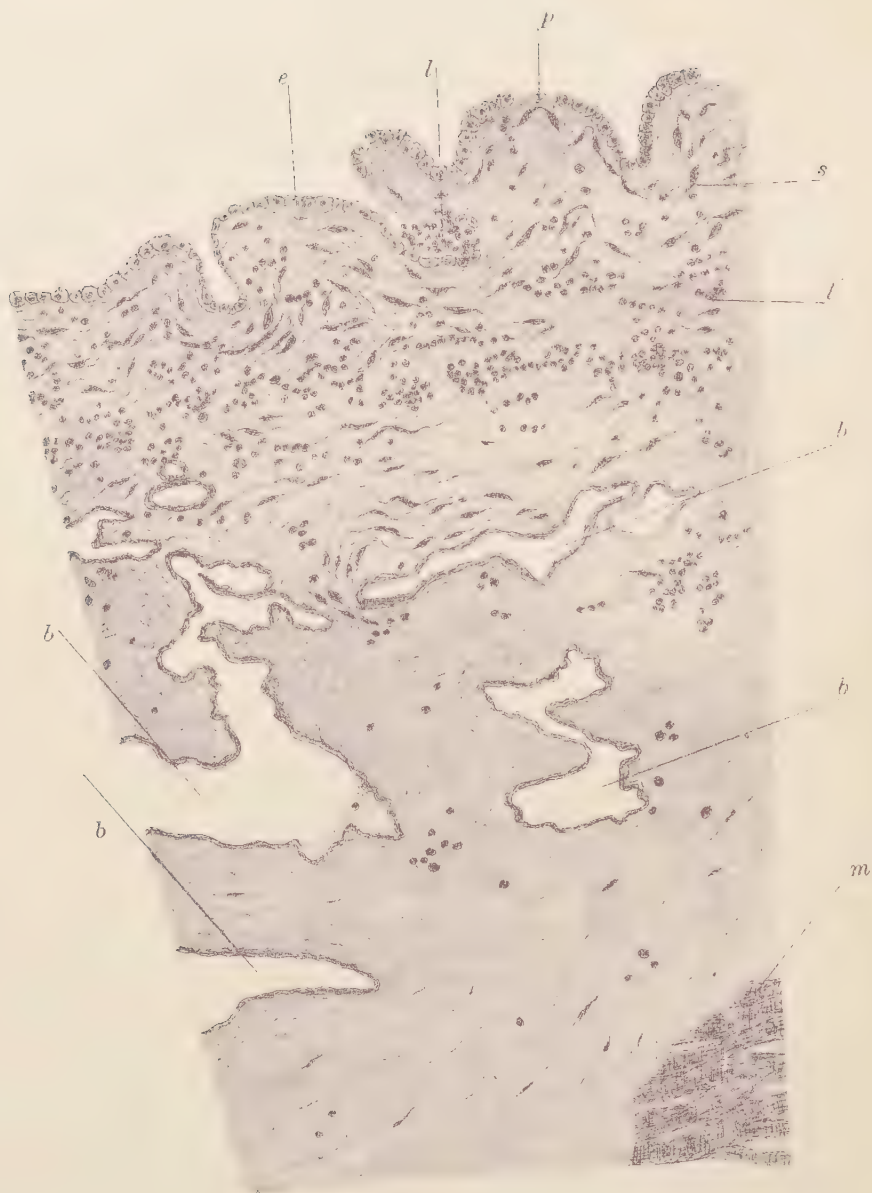


FIG. 157.—Section through the thickened pericardium, the result of old pericarditis, magnified about 150 diameters.

The superficial parts of the pericardium contain numerous spindle cells arranged parallel to the surface, which is covered with a beautiful layer of epithelium, much rounder in shape and more distinct than that covering the healthy pericardium.

p, surface of pericardium from which the epithelium has become detached; *e*, epithelium; *l*, leucocytes lying in a depression on the surface; *s*, spindle cells; *l*, leucocytes below the layer of spindle cells; *b*, *b*, large vessels; *m*, muscular fibres of the heart.

In secondary cases of pericarditis therefore, the clinical picture is made up, or, more correctly, it *may be* made up (for as we shall presently see, the symptoms due to the inflammation of the pericardium itself are sometimes entirely absent) of:—

A. Symptoms due to the primary disease.

B. Symptoms due to the pericardial inflammation itself, viz.:—

(1.) General symptoms due to the pericardial inflammation, such as fever.

(2.) Cardiac symptoms proper. Under this head we have to consider:—

(*a.*) The ‘subjective cardiac sensations,’ or the symptoms which result from the derangement of the heart as a sensitive, vital organ.

(*b.*) The symptoms which may result from the derangement of the cardiac pump—the mechanical symptoms, as I am in the habit of terming them.

(*c.*) The symptoms, which sometimes result from the pressure of the enlarged (distended) pericardial sac upon surrounding parts (*i.e.* pressure symptoms).

(*d.*) Accidental symptoms due to complications, or to the extension of the inflammatory process to adjacent parts.

Symptoms due to the primary disease.—It would be out of place here to describe in detail the symptoms which characterise the many different diseases with which inflammation of the pericardium may be associated. I must, however, emphasise the importance of looking out for the symptoms, and more especially for the physical signs of pericarditis, in all of those diseases, such as acute rheumatism, Bright’s disease, etc., in which we know that inflammation of the pericardium is apt to occur. A trivial pain over the præcordium, for instance, which under ordinary circumstances, and in many diseased conditions, might be almost ignored, should, in a case of acute rheumatism or Bright’s disease, excite grave apprehension, and necessitate minute and careful examination of the heart. And this statement is all the more necessary because of the fact, that the symptoms due to the

primary disease (notably so in acute rheumatism) are often very striking, and are apt to overshadow and obscure the symptoms, due to the inflammation of the pericardium—symptoms which are in many cases slight.

Symptoms due to the pericardial inflammation itself.—The nature and gravity of the symptoms, which depend upon the inflammation of the pericardium itself, depend upon the following conditions:—

- (1.) The extent and severity of the inflammation.
- (2.) The condition of the cardiac muscle, and of the valvular apparatus of the heart.
- (3.) The amount and character of the effusion.
- (4.) The constitutional temperament (susceptibility), and the previous state of health of the patient.
- (5.) The manner and degree in which the cardiac nerves and ganglia, on the surface of the heart and around the root of the aorta, are affected by the inflammatory process.

In those cases in which the inflammation is not very severe; in which the cardiac muscle is not implicated; in which the amount of effusion is small; in which the patient was in fair health previous to the attack; and in which he is not of an unduly susceptible temperament, the symptoms may be extremely slight or altogether absent. Indeed, from the frequency with which we find pericardial thickenings and localised adhesions after death, it is probable that many cases of this description pass entirely unnoticed. The case, which I have narrated on page 298, shows that even in severe and fatal cases of pericarditis, the symptoms may be very slight.

General symptoms due to the pericardial inflammation—fever.—The fever is, as a rule, slight or moderate, and presents no characteristic features. The tongue, bowels, and urine present the usual alterations which are met with in fever. When the pericarditis occurs in the course of a febrile disease, such as acute rheumatism, the increase of fever due to the inflammation of the pericardium is seldom sufficiently great to arrest attention. In purulent pericarditis the fever may be high, and present the irregular ups and downs which, when

associated with rigors and sweatings, are so characteristic of suppuration. When the inflammation extends to the pleura, and more especially to the lung itself, the fever is proportionately increased. In some cases there is hyperpyrexia,¹ and in these cases grave cerebral symptoms, the exact nature of which I shall presently describe, are usually developed.

The frequency of the pulse is, as a rule, considerably increased, the amount of increase in the earlier stages being proportionate to the amount of fever. In exceptional cases the pulse may be slower than normal. In the earlier stages it is sometimes full and bounding, and of high tension, but more frequently irritable and soft. In all cases of pericarditis there is a tendency to failure of the pulse and to the production of diastolic murmurs; and this is more especially the case where the myocardium is implicated. When endocarditis and valvular complications are present, the characters of the pulse may, of course, be profoundly modified, the exact characters depending upon the nature of the valvular complication.

Cardiac symptoms proper.—Subjective cardiac sensations, which result from derangement of the heart as a vital, sensitive organ, such as pain in the region of the heart, a feeling of tightness or constriction about the chest, and (occasionally) palpitation or intermittent action of the heart, are experienced in the great majority of cases of pericarditis.

Cardiac symptoms of this description are suggestive of the presence of pericarditis, but they are not distinctive of that condition. In short, the student should distinctly understand that the symptoms of pericarditis (*i.e.* the symptoms as distinct from the physical signs) are by no means characteristic.

According to Sibson's elaborate statistics, pain of a more or less continuous character is present in three-fourths of the

¹ The exact cause of the hyperpyrexia is not known. It is much more common in cases of rheumatic fever, complicated with pericarditis, than in cases which are unattended with that condition. The statistics of the Clinical Society of London seem to show that the pyrexia is not associated so frequently, as was formerly supposed, with suppression of perspiration.

cases of rheumatic pericarditis. It is most marked in cases of 'dry' pericarditis, and usually disappears when the effusion becomes copious. Its intensity is not as a rule great, but in some cases 'the suffering and distress over the heart are so great as to drown all other complaints.' In a considerable proportion of cases there is pain and tenderness in the pit of the epigastrium; and in some cases there is also tenderness on pressure over the region of the heart itself.¹

In exceptional cases a constrictive pain in the chest, radiating to the left shoulder and down the left arm, and resembling the pain of angina pectoris, is met with. It probably depends upon the fact that the cardiac nerves (branches of the cardiac or coronary plexuses) are irritated by the inflammatory process.

One of the most marked cases of angina pectoris which I have ever seen, was attended with, and apparently depended upon pericarditis. The following is an abstract of it:—

Case—Angina pectoris; pericarditis of obscure origin.

The patient was a ship-captain, æt. 49, of a florid complexion and sanguine temperament. He had suffered from syphilis, and had perhaps indulged somewhat too freely in alcohol, but had been in vigorous health up to the occurrence of the attack. The pain commenced gradually in the region of the heart, and continued more or less constantly for a fortnight. It was severe throughout the attack, and was, as a general rule, confined to the region of the heart; every now and again—some days several times in the twenty-four hours—it became greatly intensified, and assumed the characteristic features of angina pectoris. During the earlier days of the attack nothing of importance was detected in the condition of the heart, but well marked pericardial friction subsequently developed. At the end of a fortnight the pain and pericardial friction

¹ Sibson concludes, probably with truth, 'that the pain on pressure below or at the side of the ensiform cartilage is in these cases due, not to peritonitis, but to inflammation of the fibrous structure and pericardial or inner surface of the central tendon of the diaphragm, where it forms the floor of the pericardial sac, and the lower and anterior portion of that sac.' The same authority states, 'that if the pain over the heart is increased or excited by pressure over the region of the organ, it may, with an approach to certainty, be attributed to inflammation of the pleura, especially if the pain on pressure is complained of, not before, but at the time of or after the first presence of the friction sound.'—Russell Reynold's *System of Medicine*, vol. iv. pp. 230 and 224.

both disappeared, and the patient apparently made a complete recovery. I am unacquainted with the after progress of the case, and I have never been quite able to satisfy myself as to the exact cause of the attack. The aortic second sound was throughout the attack, and also subsequently, somewhat accentuated, and I have often surmised that the cause of the pericarditis was the pressure of an aneurism originating immediately above the sigmoid valves.

Mechanical symptoms, which result from disturbance of the action of the cardiac pump (such as pallor or lividity of the countenance, irregularity and failure of the heart's action, the *pulsus paradoxicus*, faintness, difficulty of breathing, orthopnoea, etc.) are usually observed in severe cases. They may be due to one or other of the following conditions:—

(a) Effusion into the sac of the pericardium sufficiently copious to embarrass the action of the heart, and to interfere with the course of the circulation. A large effusion, more especially if it is rapidly poured out, may seriously interfere with the course of the circulation, by compressing the thin-walled auricles, the *venæ cavæ*, and pulmonary veins, and so prevent the due supply of blood to the aorta and pulmonary artery. The right auricle and superior cava, in consequence of the comparative thinness of their walls, are affected most by the pressure. In consequence of the pressure on the superior cava, the veins in the neck become distended, and the greater the pressure on the superior cava the greater the distention of the veins. In cases of pericarditis, fulness of the veins of the neck is, therefore, suggestive of copious effusion, more especially when there is no evidence of endocarditis or over-distention of the right cavities of the heart.

(b.) Inflammation of the cardiac muscle.

(c.) Endocarditis and associated valvular lesions.

Mechanical symptoms are only seen in severe cases of pericarditis (cases with copious effusion), or in those cases in which the inflammation of the pericardium is associated with disease of the muscular walls or valvular mechanism of the heart. In fact, in any case of pericarditis in which mechanical symptoms were prominent, we would probably be

correct in concluding that there was something more than mere inflammation of the pericardium present (*i.e.* that either myocarditis or endocarditis was present.)

Pressure symptoms.—When the effusion is very extensive, the greatly distended pericardial sac may press upon the œsophagus and produce difficulty in swallowing (dysphagia), or upon the trachea or left bronchus, and cause cough and difficulty in breathing. These symptoms are aggravated by the recumbent, and relieved by the sitting posture.

Accidental symptoms, due to the extension of the inflammation to surrounding parts (other than the myocardium or endocardium), or to the presence of complications, are of common occurrence. In a considerable proportion of cases, pleurisy, with its attendant symptoms and signs (pain in the side, etc.) develops. In a few cases, pneumonia or pulmonary apoplexy is met with.¹ In rare cases, more especially in rheumatic pericarditis, cerebral symptoms and hyperpyrexia occur. Hyperpyrexia is, according to the elaborate report of the Clinical Society of London, much more frequent in males than in females, and is much more common in a first than in subsequent attacks of rheumatic fever. The nervous symptoms met with in connection with it are in the order of their relative frequency as follows:—‘delirium (in all phases), insomnia, restlessness, muscular tremors, involuntary discharges, subsultus tendinum, coma (a late symptom), headache, tremor of tongue, deafness, tonic spasms (in two cases of tetaniform character), risus sardonicus, convulsions, floccitation, tinnitus aurium, giddiness, drowsiness, vomiting, silliness of manner, fearfulness, hesitating speech, chorea, hyperæsthesia.’ ‘The delirium, which is the most frequent symptom met with in these cases, sometimes precedes, sometimes

¹ Pleuritic pain was present in one half of Sibson's cases of rheumatic pericarditis. He says that it may be due to two causes, viz.:—one, the extension of the inflammation through the fibrous structure of the pericardium to the pleura covering it; the other, the occurrence of pulmonary apoplexy with its attendant pleurisy.—*Russell Reynolds*, vol. iv. p. 233.

accompanies, and sometimes follows the onset of the hyperpyrexia.' One half of the cases of hyperpyrexia, collected by the Clinical Society, terminated fatally. The duration, after the development of the nervous symptoms, varies from a few hours to three, four, or five days.

In addition to the delirium which is associated with hyperpyrexia, Sibson, Austin Flint, and others, have described a peculiar form of mental derangement, characterised by a state of taciturn melancholia and (according to Austin Flint) a suicidal tendency. According to Sibson's observations, the cases of pericarditis, in which this peculiar form of mental aberration was met with, were almost always complicated with endocarditis, and in some cases chorea subsequently developed; and Sibson suggests that the condition is due to embolic plugging of minute vessels of the cerebral cortex. This form of delirium differs from the delirium which is associated with hyperpyrexia, in as much as it is not associated with fever, its duration is much longer (from three weeks to three months, according to Sibson), and in the fact that the majority of cases, which are not complicated with chorea, terminate in recovery.

In a third group of cases of pericarditis, a form of delirium which resembles more or less closely delirium tremens, is met with. This form is, according to Sibson, usually associated with moderate fever, and occurs in persons of an anxious nervous disposition, in those who have been addicted to alcoholic excess, or who have been exposed to want and privation. It is sometimes seen in connection with hyperpyrexia.

In addition to the symptoms already enumerated, the following may be added:—

Alteration of the countenance.—In a large proportion of cases the facial physiognomy is altered. In some the face is flushed.¹ This condition is seen more especially in the earlier stages of rheumatic cases (*i.e.* in cases in which the

¹ Sibson suggests that flushing and pallor of the countenance may in some cases be due to reflex disturbance of the vaso-motor centre, the pericardial inflammation acting as the peripheral source of irritation.

action of the cardiac pump is not as yet embarrassed), and is generally associated with profuse sweating.

In others, the face is pale, pinched, and anxious; and there may be other indications of collapse and nervous exhaustion, such as nausea, vomiting, restlessness, insomnia, a weak thready or irregular pulse, etc. Severe pain in the region of the heart or in the joints, and sudden failure of the heart's action, as the result of copious effusion, myocarditis, valvular complications, etc., are the most frequent causes of this state of depression.

In others, again, the countenance is more or less dusky, livid, or congested. This condition is generally seen in the later stages of the case, and is associated with obstructed venous return, the causes of which I have already described in speaking of the mechanical symptoms. (See p. 311.)

Cough, hoarseness, and aphonia.—A short, dry, irritable cough is often present, and in some cases there is hoarseness or aphonia. The latter symptoms probably depend upon irritation of the left recurrent laryngeal nerve as it winds round the root of the aorta.¹

Physical signs.—The physical condition of the pericardium is ascertained by the same means by which we ascertain the condition of the heart itself, and it is important to remember that, under normal circumstances, the opposed surfaces of the sac are in close contact, and move smoothly and noiselessly one upon the other, and that the signs derived from the physical examination of the pericardium are entirely negative. In other words, the size and shape of the pericardium correspond, in conditions of health, exactly to the size and shape of the heart itself, and the outlines of the two structures cannot be distinguished by means of percussion, while on listening over the pericardium the sounds of the heart are alone heard.²

¹ In a case observed by Baeumler (quoted by Bauer, Ziemssen's Cyclopædia, vol. vi. p. 603), double paralysis of the vocal cords, due to the pressure of a large pericardial effusion upon both recurrent nerves, was observed.

² It has been stated that, during violent action of the heart, a friction sound is sometimes produced *within* the normal pericardium, but in common with other observers I am disposed to doubt the statement.

Now in pericarditis the physical conditions are entirely altered, and it is only by keeping the nature of the alterations prominently in view, that it is possible to understand intelligently the alterations in the physical signs which occur when the membrane is inflamed. These alterations are as follows:—

Firstly, the surfaces of the pericardium become covered with a layer of lymph, and the rubbing together of the two opposed rough and inflamed surfaces (*i.e.* of the visceral and parietal layers of the sac), which occurs during the systolic and diastolic movements of the heart, is attended with the production of vibrations, which are heard, through the stethoscope as *friction-sounds* or murmurs, and can, in some instances, be felt by the hand placed over the præcordium as *friction fremitus*.

Pericardial friction-sounds and pericardial friction fremitus may be termed *the physical signs which result from roughening of the membrane*.

Secondly, fluid is poured out into the interior of the sac. In many cases the distention of the sac is sufficiently great—

(*a*) To give rise to increased dulness on percussion over the præcordium, and to enable us, in *some* cases, to differentiate, by means of percussion, the outline of the pericardium from the outline of the heart.

(*b*) To produce such displacement in the position of the heart, that the cardiac impulse and the apex beat are either altered from the normal position or (in rare cases) completely effaced—physical facts which we determine by means of palpation.

(*c*) To cause lateral displacement of the lungs, and downward displacement of the diaphragm and therefore of the left lobe of the liver.

(*d*) To cause, more especially where the effusion is considerable and the chest wall elastic, bulging of the præcordium and widening of the intercostal spaces corresponding to this part of the chest.

(*e*) By compressing the auricles and veins, to cause distention of the veins of the neck, and to interfere with the natural

course of the circulation—effects which I have already alluded to, and which are of course manifested by distinct symptoms and physical signs.

The physical signs which are due to the conditions enumerated under headings *a*, *b*, *c*, *d*, and *e*, may be termed the *physical signs which result (or rather which may¹ result) from effusion into the sac*.

Now in some cases the effusion is never sufficiently great to produce increased dulness on percussion, and the other physical signs which I have termed ‘the physical signs which result from effusion into the sac.’ In other cases, when the patient first comes under observation, the effusion is not as yet sufficiently extensive to alter the percussion outline of the heart, although it subsequently may become so. In others again, although sufficiently extensive to produce increased dulness at an earlier period of the case, it has already been in great part absorbed before the patient comes under observation. In all of these cases the physical signs will simply be those which are due to *roughening* of the walls of the sac, in short, those of the so-called *dry*² form of pericarditis.

In the so-called *moist* variety of pericarditis, on the contrary, the physical signs are not merely those which result from *effusion into the sac*. The pericardial friction usually continues throughout the period of effusion. Moist pericarditis is, therefore, characterised by physical signs resulting both from effusion into the sac and roughening of the pericardial membrane.

Let us now consider some of the more important of these physical signs a little more in detail.

Character of Pericardial Friction Sounds.

Rhythm.—In typical cases pericardial friction is double, a *to-and-fro* sound, as it has been termed, the *to* and *fro* corre-

¹ I say *may* result, for unless the effusion is considerable, some of them at all events (distention of the veins of the neck, for instance), are not produced.

² The somewhat artificial division into *dry* and *moist*, is especially useful in describing the physical signs. Dry pericarditis may be defined as pericarditis in which the amount of effusion is *not* sufficiently extensive to be detected by means of percussion; while *moist* pericarditis may be defined as pericarditis in which the effusion *is* sufficiently extensive to be detected by means of percussion.

sponding to the rubbing of the two rough surfaces of the membrane during the systole and the diastole of the heart respectively. The synchronism with the heart sounds is seldom very exact, and in this respect there is, as we have previously seen, an important distinction between pericardial and endocardial murmurs. The two divisions of the sound are, as a rule, of equal duration, the systolic portion being generally louder than the diastolic. Occasionally the pericardial friction sound is single, and it is then systolic (*i.e.* it corresponds rather to the first than to the second sound of the heart). In some cases it is triple, and presents a *presystolic* as well as a systolic and diastolic rhythm.

Point of maximum intensity and direction of propagation.—Pericardial friction is usually best heard over that part of the heart which comes in most direct and forcible contact with the front wall of the chest. Under ordinary circumstances, therefore, the friction is first and best heard over the front of the right ventricle, that is to say, over the lower end of the sternum and the adjacent costal cartilages. In many cases the friction sound is limited to the præcordial region, and very often to the part of the chest wall which corresponds to the exact point of the sac at which the friction is produced; though, as Sibson has shown, when the vibrations are directly communicated to the sternum, as they are most likely to be in cases of dry pericarditis (*i.e.* when there is no intervening layer of lung tissue or of fluid between the point of their production and the front wall of the chest), the friction sound may be conducted in various directions by the sternum and costal cartilages, which are attached to it—the sternum acting as a sounding board. Pericardial friction sounds have no definite and distinct lines of propagation as endocardial murmurs have.

Sound Character.—In the majority of cases the sound, as the term *friction* murmur suggests, is harsh, and gives one the distinct impression of being produced by the rubbing together of two rough surfaces. In many cases it is actually grating or creaking (resembling the sound produced by the creaking of new leather '*bruit de cuir neuf*'); exceptionally it is soft in

character. It is usually superficial, *i.e.* it seems to be produced immediately under the ear. In addition to these characters, which are usually of themselves quite conclusive, pericardial friction is almost invariably intensified, and in many cases its sound characters are absolutely altered by the pressure of the stethoscope; the effect of this pressure being of course to bring the two roughened surfaces of the pericardium in closer and firmer contact one with the other. Pericardial friction sounds, too, are very apt to undergo spontaneous alterations in character (rhythm, tone, area, etc.), and this is more particularly the case in the earlier periods of the inflammation, and depends of course upon the fact that the physical conditions within the sac (amount of exudation, etc.), are then undergoing rapid alterations.

When the friction sound is loud, the normal heart sounds are, as a rule, completely obscured by it; when the friction sound is soft the normal heart sounds may, as it were, be heard through it. In those cases in which endocarditis and valvular complications are present, valvular murmurs are sometimes audible in addition to the pericardial friction sounds.

The loudness and tone of the pericardial friction sound seem to depend upon:—(1) the force with which the two opposed surfaces of the roughened pericardium are rubbed together, and the amount of movement which takes place between them, that is to say upon the force with which the heart is contracting, and the amount of the external resistance opposed to its contraction, which in its turn depends upon the position of the organ in the thorax, and especially the relationship of the organ to the front wall of the chest; and (2) the amount and consistency of the lymph. Vigorous action of the heart; close contact of the organ with the front wall of the chest, more especially of the right ventricle with the sternum; a small amount of fluid in the sac; and a thick layer of tolerably tough lymph on the two opposed surfaces of the pericardium, seem to be the conditions which favour the production of loud pericardial friction; and other things being equal, the louder the friction sound the more extensive the area over which it is propagated.

Dulness on Percussion over the Præcordium.

The dulness which is due to effusion into the sac of the pericardium presents certain important characteristics.

Its *extent* depends upon the amount of effusion into the sac, the more extensive the effusion the greater the dulness. The condition of the anterior margins of the lungs is also of considerable importance ; when the lungs are very voluminous, as in emphysema, or when their anterior margins are fixed to the front wall of the chest by adhesions, the dulness is by no means so extensive as when they are normal and can be readily pushed aside and compressed by the enlarging sac.

The *form* and *outline* of the dulness correspond to the form and outline of the pericardial sac ; and since the pericardium is not reflected at the base of the heart, but includes in its narrower, upper part a considerable portion of the great vessels, it follows that the area of dulness which results from distention of the sac is pyramidal (more correctly a truncated pyramid, the apex being above, the base below) or pear-shaped—the stalk of the pear corresponding to the upper and narrower part which surrounds the great vessels, the body of the pear to the lower part of the distended sac which surrounds the heart itself. The peculiar shape of the dulness is of great diagnostic value ; and another point which is equally important as an indication of pericardial effusion, but which unfortunately is not always present, is that in some cases the area of dulness extends beyond, *i.e.* further to the left than the apex-beat—*i.e.* the left apex beat. In figure 158, the shape of the pericardium, when artificially distended with fluid, is shown ; in fig. 159, the appearance which the sac may present when greatly distended in disease. In fig. 160, the characteristic pear-shaped outline, which the dulness presents, is well seen.

Some recent writers differ from the usually received opinion, that the form of the area of cardiac dulness is characteristic of pericardial effusion. Thus Dr Rotch, in the Supplement to Ziemssen's *Cyclopædia of the Practice of Medicine*, page 363, says, 'Bauer's opinion, that the triangular shape of the area of dulness depends upon the shape of the pericardial sac, has not been substantiated by later observations, the theory of Duchek having been found to be more correct, namely that in cases uncomplicated by

pleuritic adhesions it is the retraction of the edges of the lungs which determines the shape of the absolutely dull area and that therefore the so-called triangular figure can be produced by an enlarged heart as well as a pericardial effusion.'

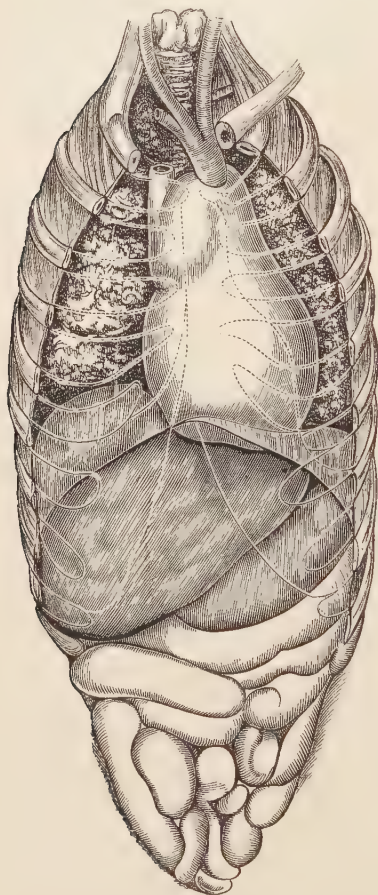


FIG. 158.

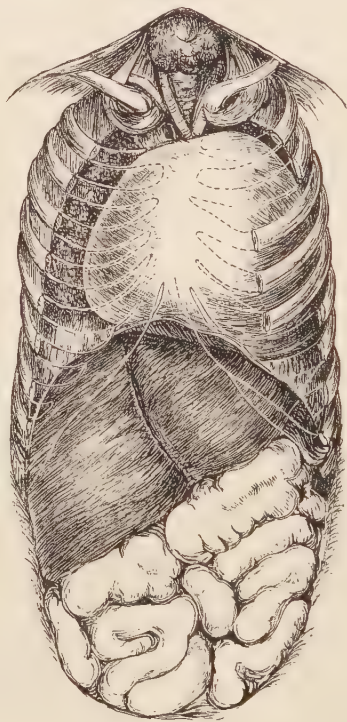


FIG. 159.

FIG. 158.—Pericardium distended into fifteen ounces of fluid. (After Sibson.)

FIG. 159.—Case of pericarditis in which the sac contained $3\frac{1}{4}$ lbs. of fluid.

Altered Position of the Heart and Apex Beat.

The effusion at first collects at the sides of the heart, at the bottom of the pericardial sac, and around the great



FIG. 160.—Outline of percussion dulness in a case of pericardial effusion.
(After Sibson.)

vessels. As it becomes more and more copious the sac becomes, of course, more and more distended, and the heart is pushed upwards, the apex being tilted towards the left. In consequence of this alteration in the position of the heart, the position of the cardiac impulse is altered, the lower boundary of the impulse being situated in the fourth or even the third, instead of in the fifth interspace, as it is in health. The apex beat not unfrequently corresponds to the left nipple, and in some cases it may be situated slightly above, and to the outer side (*i.e.* to the left) of the nipple. When the effusion is copious, the apex beat is usually impaired in force or altogether effaced. In exceptional cases, owing to the fact that the heart is enlarged, as the result of previous disease, or tied down, as the result of previous pericarditic adhesions, the position of the heart and apex beat is not altered. When the effusion is absorbed, the heart and with it the cardiac impulse and apex beat usually return to the normal position. An exception to this, the general rule,

occurs in some cases in which adhesions are formed while the heart is still in its displaced position.

In consequence of the alteration in the position of the heart, which occurs in most cases of copious effusion, the area of the pericardial friction sounds, which we have previously seen persist, as a rule, through the period of effusion, is also displaced upwards. The to-and-fro murmur may, therefore, be heard in the third or fourth interspaces, and considerably to the left of the sternum instead of over the lower end of the sternum and the adjacent lower left costal cartilages, as it is in cases of 'dry' pericarditis. When the fluid is absorbed, and the heart returns to its normal position, the area of friction moves downwards with it.

Prominence or bulging of the præcordium and swelling in the pit of the epigastrium are sometimes observed when the effusion is copious. Decided prominence of the præcordial region only occurs when the chest wall is elastic, and is therefore most commonly seen in young subjects; it would be more frequent in women than in men if it were not usually obscured by the prominence of the left mamma. The lower left intercostal spaces are widened, and some observers have described fluctuation in them, but this condition (fluctuation) is probably very rarely (if ever) present, in acute pericarditis.

General survey of the symptoms and physical signs.—Let us now take a brief general survey of the symptoms and signs of acute pericarditis; and remembering that acute inflammation of the pericardium is usually secondary, let us omit for the purposes of description the symptoms which are due to the primary disease, and limit the description to those which result from the inflammation of the pericardium itself.

It will be self evident from what I have already stated, that cases of acute pericarditis vary very considerably in the severity of the symptoms, and in the nature of the physical signs which they present. The following types of the disease may be described:—

1. *Cases of latent pericarditis.*—In these cases the symptoms are extremely slight or altogether absent, and in many

cases of this description the condition entirely escapes notice. The effusion is for the most part slight, but the case which I have related on page 298, shows that a considerable effusion may be unattended by any marked symptoms. Latent pericarditis very rarely indeed proves fatal; the case to which I have just alluded shows, however, that death does occasionally occur from syncope.

2. *Uncomplicated cases of dry pericarditis which are attended by distinct symptoms.*—In these cases there are no mechanical symptoms; pain in the region of the heart is the chief complaint; the countenance may present evidence of suffering, but the general constitutional symptoms are not, as a rule, severe. On auscultation the characteristic pericardial friction murmur is heard over the præcordium (usually over the lower end of the sternum and adjacent costal cartilages, though in some cases it is much more diffused); there is no distinct increase of the percussion dulness; friction fremitus is occasionally felt over the præcordial region.

3. *Cases of moist pericarditis in which the effusion is not sufficiently copious to seriously embarrass the action of the heart, and in which there is no myocarditis or endocarditis.*

In this group, which includes a large number of the cases of acute pericarditis met with in practice, subjective cardiac sensations due to derangement of the heart as a sensitive, vital organ (such as pain, constriction of the chest, palpitation, etc.), are present, but mechanical symptoms (see page 311) are either slight or altogether absent. The physical signs vary with the stage of the disease. Pericardial friction is the first physical sign which can be observed; in a short time, usually in a few hours, dulness on percussion is developed and gradually assumes the characteristic outline, which I have previously described in detail (see page 319); when the effusion is copious the whole cardiac impulse is raised, the apex beat displaced upwards and to the left, the force of the apex beat being usually impaired and sometimes altogether effaced; with the elevation of the heart the area of pericardial friction is also elevated; in young subjects the præcordial region may be more prominent than in health. As the

effusion is absorbed, the area of cardiac dulness gradually diminishes, the apex beat regains its normal position, and the friction murmurs, which during the period of effusion are, as a rule, strictly limited to the area of dulness, and usually to the area of the cardiac impulse, may become more diffused, and are sometimes heard over the whole of the sternum and adjacent parts of the chest. In exceptional cases friction fremitus may now be felt when the hand is placed over the præcordium.

4. *Cases of moist pericarditis in which the effusion is sufficiently copious to seriously embarrass the action of the heart, and cases of pericarditis (either dry or moist) which are complicated, with symptoms due to myocarditis or to endocarditis.*

The cases included under this group are attended with grave symptoms indicative of serious interference with the course of the circulation, and with serious failure of the action of the heart, such as marked pallor or lividity of the countenance, faintness, extreme exhaustion, an unusually rapid, weak, irregular, and dicrotic pulse, considerable distention of the veins of the neck, considerable dyspnœa or orthopnœa, dropsy, etc. The physical signs up to a certain point are the same as those described as characteristic of the third group, viz., the physical signs proper to a considerable and usually progressive pericardial effusion. When the heart begins to fail the friction murmur usually becomes diminished in intensity; and alterations in the rhythm of the heart (irregular action, etc.) may be heard on listening over the præcordium. With the occurrence of endocarditis valvular murmurs are developed. It must not, however, be supposed that valvular murmurs are only heard in those cases of pericarditis which are included under this group. In a large proportion of the cases of pericarditis (of the rheumatic cases at all events) which have been included in groups 1, 2, and 3, endocardial murmurs are also present. In this group I include those cases in which in addition to the valvular murmurs, *symptoms* due to valvular lesions are present, *i.e.* when the valvular lesion is sufficiently severe to mechanically interfere with the course of the circulation.

Onset, Course, and Termination.

Onset.—The onset is in many cases insidious, pain and discomfort in the region of the heart being very generally the first symptoms which attract attention. In other cases, the attack is ushered in by a feeling of chilliness and by the usual malaise, etc., which so frequently attend the commencement of febrile diseases, very exceptionally there is a distinct rigor. In some cases nausea and faintness are the first symptoms.

Course.—In acute cases the inflammation of the pericardium rapidly runs its course,—a fact which explains the frequent alterations in the physical signs which are met with in the disease. In the course of three or four days the effusion has usually reached its height; the period during which the sac remains fully distended, is, as a rule, brief, in most cases absorption begins to occur within a few hours after the effusion has reached its highest point. Unless the case should become chronic, which it only does in exceptional cases, the period during which the effusion is stationary, so to speak, is seldom longer than two or three days. The period of absorption and resolution generally occupies several days. The whole duration of the attack, from the commencement to the disappearance of friction, is usually from one to three weeks, though many exceptions to this rule occur, in some cases the duration is shorter, in others much longer than that specified.

Termination.—The natural termination of inflammation of the pericardium, is recovery with the formation of adhesions *i.e.* with partial or total obliteration of the sac. Occasionally, but very rarely in cases of rheumatic origin, death occurs during the acute stage; in a few cases the condition becomes chronic. But to these points I shall again refer in speaking of the prognosis.

Diagnosis.—In considering the diagnosis of pericarditis, it is essential to remember that the symptoms are only suggestive but not distinctive; and that inflammation of the pericardium can only with certainty be recognised by means of the physical signs. Friction sounds, synchronous with the action

of the heart, and increased dulness on percussion of the form and outline, which have been previously described, are the physical signs of greatest importance ; but neither of them is, when taken singly, absolutely conclusive, for on the one hand there are certain conditions in which sounds are heard resembling those produced in an inflamed pericardium, and on the other, there are several conditions in which dulness on percussion, resembling more or less closely the dulness due to pericarditis, is present.

Steps in the diagnosis of pericarditis.—When a case of supposed pericarditis comes under observation, we have therefore to determine :—

1. Is inflammation of the pericardium actually present ?
2. If the case is one of pericarditis, what is the cause of the condition, and what is the character of the effusion ?
3. Is the case complicated with myocarditis or endocarditis ?

Step No. 1. Is the case one of pericarditis ?—In some cases it is easy to decide this point, in others difficult.

When a patient, who has been previously in good health, is attacked with an acute illness, more particularly with one or other of those affections, in the course of which acute pericarditis is apt to occur ; and when a to-and-fro friction murmur and increased dulness on percussion, are present over the præcordium (more especially when the increased dulness on percussion is rapidly developed under, as it were, the eye of the observer), the diagnosis is self-evident. In other cases the diagnosis is difficult ; and it will be necessary to consider in the next place, the conditions in which sounds resembling pericardial friction, or dulness resembling, more or less closely, that produced by pericarditis is present ; and the means by which they are to be distinguished.

The differential diagnosis of pericarditis with friction, and the other conditions with which it is likely to be confounded.

The typical to-and-fro friction murmur of pericarditis is of course, synchronous with the action of the heart, and it is

by this means at once distinguished from ordinary pleuritic friction. Cases are, however, occasionally met with in which a pleuritic murmur is of cardiac rhythm. In the *first* place, therefore, we must consider :—

The differential diagnosis of pericardial friction and pericardial-pleural friction.

In some cases it is impossible to pronounce a positive opinion, but the distinction can generally be made by attention to the points detailed in the following table :—

TABLE I.

The Differential Diagnosis of Pericardial Friction and Pericardial-pleural Friction.

	Pericardial Friction.	Pericardial-pleural Friction.
<i>Where audible.</i>	Usually heard over the centre of the right ventricle.	Usually heard over the borders, more especially the left border, of the heart.
<i>Effect of respiration.</i>	Seldom much affected by the respiratory movements, never completely arrested by holding the breath. Never actually* converted into ordinary friction by a full inspiration or deep expiration.	Always more or less affected by the respiratory movements; often completely arrested on holding the breath; often converted into ordinary pleural friction by a full inspiration or deep expiration.

* *Note* —This point is not of much importance, for true friction is not unfrequently accompanied by pleurisy, and in cases of this description the pericardial friction may on a full respiration, be masked (though it is not actually replaced) by the ordinary friction sound.

In the *second* place, pericardial friction is sometimes soft in character (*i.e.* it lacks the harsh characters which friction sounds typically possess); and since a double murmur is very frequently generated at the aortic orifice, we must now consider the differential diagnosis of the double murmur which is due to pericarditis on the one hand and to combined aortic stenosis and incompetence on the other. The points of distinction are given in table II.

TABLE II.

The Differential Diagnosis of the Double Murmur due to Pericarditis, and the Double Murmur due to Aortic Stenosis and Incompetence.

	Double Murmur of Pericarditis.	Double Murmur of Aortic Stenosis and Incompetence.
<i>Position of maximum intensity.</i>	Usually loudest over the right ventricle and lower half of the sternum.	Although the diastolic portion of the murmur is often heard loudest over the lower end of the sternum, it is also well heard at the base of the heart.
<i>Area where audible, and direction of propagation.</i>	Usually heard over a limited area, and not propagated over the course of the aorta, and into the vessels of the neck.	Usually heard over an extensive area, and propagated over the course of the aorta, and (usually) into the great vessels of the neck.
<i>Relationship to the heart sounds.</i>	Does not replace but only obscures (more or less completely) the heart sounds; and is not exactly synchronic with them.	Does actually replace (but not always completely) the heart sounds, and is always exactly synchronic with them.
<i>Variability.</i>	Apt to vary from day to day, or from hour to hour, both in respect to its position and sound characters; the interval between the systolic and diastolic portions is not always of the same duration, and not always distinct.	Remains constant as regards its position and sound characters; the interval between the systolic and diastolic portions is always of the same duration and is distinct.
<i>Effects of external pressure.</i>	Almost invariably altered in character by the pressure of the stethoscope, when soft is often converted into a rough friction sound.	Uninfluenced by the pressure of the stethoscope.
<i>Pulse.</i>	Not jerking, visible, collapsing or tortuous.	Is jerking, visible, collapsing, and often tortuous.
<i>Condition of the left ventricle; outline of dulness; force and position of apex beat.</i>	Not much altered in size unless previously diseased. Dulness, pear-shaped with well-marked stalk. Apex beat feeble or effaced, tilted upwards and to the left.	Almost invariably hypertrophied and dilated—Dulness, heart-shaped without well-marked stalk. Apex beat strong, displaced downwards and to the left.
<i>Ætiology.</i>	Usually developed acutely in the course of acute rheumatism, Bright's disease, etc.	May be developed acutely as the result of rheumatic or septic endocarditis, but is usually developed very gradually as the result of atheroma.

In the *third* place a pericardial friction murmur is sometimes (instead of being double) single and systolic ; and in such cases it may be mistaken for a murmur generated within the heart itself, *i.e.* a systolic mitral, tricuspid, aortic, or pulmonary murmur. These are the cases in which pericarditis is liable to be mistaken for endocarditis ; the mistake is most likely to be made when the pericardial murmur is soft in character.

In table III., p. 330, I have detailed the points of distinction between typical pericarditis and typical endocarditis, while in table IV., p. 331, is given the differential diagnosis of those cases of pericarditis, in which the murmur is single, systolic, and soft, and of the mitral, tricuspid, aortic, and pulmonary murmurs which are present in cases of chronic valvular lesions, anæmia, etc.

In the *fourth* place, we must consider the differential diagnosis of increased dulness due to pericarditis and increased dulness due to other conditions. Simple serous effusion into the sac of the pericardium (hydropericardium) and enlargement of the heart itself (more especially dilatation of the heart), are the conditions included under this group which are most likely to give rise to difficulty.

The differential diagnosis of increased dulness due to pericarditis and simple dropsy of the pericardial sac.

In both of these conditions the form and outline of the dulness are the same, but the diagnosis can usually be made without difficulty, by attention to the points detailed in table V., p. 332.

The differential diagnosis of pericardial effusion and enlargement of the heart itself, is a point of considerable practical importance ; for an enlarged (more especially a dilated) heart has been more than once punctured under the impression that the case was one of effusion into the pericardial sac. The points to which attention should be directed in making the distinction are shown in table VI., p. 333.

TABLE III.

The Differential Diagnosis of Acute Pericarditis and Acute Endocarditis.

	Pericarditis.	Endocarditis.
1. <i>Characters of the murmur.</i>	In typical cases a double (to-and-fro) murmur, which obscures, but does not actually replace the heart sounds, and which is not exactly synchronic with them. Exceptionally the murmur is single and systolic.	In the majority of cases the murmur is single and systolic, occasionally double (aortic). The murmur replaces one or other of the heart sounds, and is exactly synchronic with it.
(a) <i>Rhythm of murmur.</i>		<i>Note.</i> —Exception in the case of pre-systolic murmurs, which are however rarely due to acute endocarditis
(b) <i>Sound characters.</i>	In typical cases a harsh friction sound; exceptionally, soft; superficial.	Usually soft and blowing, deep-seated.
(c) <i>Position of maximum intensity.</i>	Usually over the right ventricle, but only accidentally, having its point of differential maximum intensity in the mitral and tricuspid areas.	In the mitral, tricuspid, aortic or pulmonary areas.
(d) <i>Direction of propagation.</i>	Usually very limited in area, and not propagated in any of the definite lines of propagation of endocardial murmurs.	May be heard over an extensive area, and propagated in certain definite directions (see table VIII., p. 527).
(e) <i>Variability.</i>	Character liable to change (position, loudness, harshness, etc.,) within brief periods of time.	Character more constant.
(f) <i>Effects of pressure.</i>	Almost always intensified, and often modified in other respects, by the pressure of the stethoscope.	Not modified by pressure.
2. <i>Outline of dulness on percussion.</i>	Area of dulness often increased, and pear-shaped (see p. 321).	Dulness not, as a rule, much increased; its exact outline depends on the cavity or cavities of the heart which are enlarged.
3. <i>Impulse, apex-beat.</i>	Feeble or effaced; if much effusion apex tilted upwards and to the left.	Impulse may be strong; apex in normal position. If the left ventricle is hypertrophied, apex displaced downwards and to the left; if the right ventricle is enlarged, impulse may be in the pit of the epigastrium.
4. <i>Pain over the præcordia, and tenderness in epigastrium.</i>	Usually present.	Usually absent.

TABLE IV.—*Differential Diagnosis of the typical (single, systolic, soft), murmur of Pericarditis and of the systolic mitral, tricuspid, aortic, and pulmonary murmurs.*

	Pericarditic Murmur.	Mitral Systolic Murmur.	Tricuspid Systolic Murmur.	Aortic Systolic Murmur.	Pulmonary Systolic Murmur.
<i>Position of maximum intensity of murmur.</i>	Usually over lower end of sternum and adjacent cartilages; may be (accidentally) in mitral and tricuspid areas.	Left apex beat.	Lower end of sternum and adjacent cartilages.	Second right costal cartilage.	Second left interspace close to sternum.
<i>Direction of propagation of murmur.</i>	Usually limited; not propagated in definite lines of endocardiac murmur.	Upwards and outwards to left axilla.	Upwards and to right.	Over course of aorta and into vessels of neck.	Upwards and to left (but limited).
<i>Synchronism of murmur with first sound.</i>	Not exact.	Exact.	Exact.	Exact.	Exact.
<i>Variability of the murmur.</i>	Apt to vary.	Usually constant in character.	Sometimes varies, usually constant in character.	Constant in character.	Constant in character.
<i>Effects of pressure on murmur.</i>	Intensified and often converted into a typical to- and -fro friction murmur.	Not altered in character.	Not altered in character.	Not altered	Not altered in character.
<i>Outline of cardiac dulcness.</i>	If increased, tends to assume pear-shaped form.	That of enlarged left ventricle; in some cases also that of enlarged right heart.	That of enlarged right heart.	That of enlarged left ventricle	Usually that of dilated right heart
<i>Impulse and apex-beat.</i>	Usually feeble; if copious effusion apex tilted upwards and outwards.	Often strong; apex displaced downwards and outwards, may be pulsation in epigastrium.	Usually pulsation in epigastrium.	Usually strong; apex displaced downwards and outwards.	Impulse often in the epigastrium.
<i>Dropsy</i>	Usually absent, unless complications.	Usually present.	Usually marked.	Usually absent	May be slight.
<i>Pain over præcordia, and tenderness in epigastrium</i>	Usually present.	Usually absent.	Usually absent.	Occasionally present.	Absent.
<i>Ætiology.</i>	Acute; usually in the course of rheumatic fever, Bright's disease, etc.	May be acute, often very chronic.	May be acute, often very chronic; generally secondary to mitral lesions or lung disease.	May be acute, often very chronic.	May be acute, usually in the course of anæmia.

TABLE V.

*The Differential Diagnosis of increased Dulness due to
Pericarditis and Hydropericardium.*

	Pericarditis.	Hydropericardium.
<i>Pericardial friction.</i>	Usually present, even at the height of the effusion ; always present at some stage of the case, <i>i.e.</i> before or after the height of the effusion.	Absent.
<i>Subcutaneous dropsy and effusion into other serous sacs.</i>	Usually absent, unless complications (great venous engorgement from pressure, valvular lesions, Bright's disease, etc.)	Hydropericardium is part and parcel of a general dropsy, and usually a late event in that condition.
<i>Fever.</i>	Usually present.	Absent, unless some complication.
<i>Pain over præcordia, and tenderness on pressure in epigastrium.</i>	Usually present.	Seldom observed.
<i>Ætiology.</i>	Usually occurs in the course of acute rheumatism and cirrhotic Bright's disease, etc.	Usually in the terminal stages of mitral lesions ; or, in the course of Bright's disease, more especially the acute form, and the large white chronic variety.

The differential diagnosis of pericardial effusion and of dilatation of the heart.—The difficulty chiefly occurs, when the patient comes under observation with the pericardial dulness already developed, and more especially in those cases in which it is stationary and chronic, and when there is no pericardial friction. In both conditions (pericarditis with effusion and dilatation of the heart) there is increased dulness on percussion over the præcordia, a feeble impulse, usually feeble sounds, and often dyspnœa, venous engorgement, dropsy, etc. In order to arrive at a correct conclusion, an accurate examination of the heart, and careful inquiry into the previous history of the case, and the other points detailed in the following table are indispensable.

TABLE VI.

The Differential Diagnosis of increased Dulness due to Pericarditis and Dilatation of the Heart.

	Pericarditis with Effusion.	Dilatation of the Heart.
<i>Outline of Dulness.</i>	Dulness pear-shaped, and enlargement chiefly upwards.	Dulness not pear-shaped, and enlargement chiefly downwards.
<i>Rate of development of Dulness.</i>	Often rapid, and then characteristic.	Usually very slow; though a rapid dilatation of the heart sometimes occurs.
<i>Impulse and apex-beat.</i>	The impulse, when present, is in the 3d or 4th left interspace; apex beat tilted upwards and outwards, or effaced.	Impulse can usually be felt to the left of the lower end of the sternum, or in the epigastrium.
<i>Relation of dulness to left apex-beat.</i>	Dulness may extend to the left of the left apex-beat.	Dulness does not extend to the left of left apex beat.
<i>Auscultation.</i>	Pericardial friction may be present; when absent, sounds are obscured.	Pericardial friction not present; sounds short, but usually clear.
<i>Pain over præcordia and tenderness in epigastrium.</i>	Often present.	Usually absent.
<i>Pulsation in the veins of the neck.</i>	May be present if endocarditis complicates.	Often present when right heart dilated.
<i>Ætiology.</i>	Usually acute, in course of acute rheumatism, cirrhotic Bright's disease.	Usually chronic; often associated with chronic valvular lesions, fatty and fibroid degeneration, anæmia, etc.
<i>Fever</i>	Often present.	Absent unless from some complication.

The differential diagnosis of increased dulness due to pericardial effusion and hypertrophy of the heart, can usually be made without difficulty. (See table VII.)

TABLE VII.

*The Differential Diagnosis of increased Dulness due to
Pericarditis and Hypertrophy of the Heart.*

	Pericarditis with Effusion.	Hypertrophy.
<i>Outline of dulness.</i>	See remarks in Table VI.	See remarks in Table VI.
<i>Rate of development.</i>	Usually rapid.	Usually slow.
<i>Impulse ; apex-beat.</i>	Impulse, when present, is in the 3d or 4th left interspace, and is feeble ; apex tilted upwards and outwards, or beat effaced.	Impulse powerful ; if left ventricle hypertrophied, apex displaced downwards and outwards ; if right ventricle hypertrophied, apex displaced downwards and inwards, beat may be in the epigastrium.
<i>Pulse.</i>	Weak and quick ; may be paradoxical.	Character of the pulse depends on the side of the heart which is hypertrophied, and the cause of the hypertrophy. When left ventricle hypertrophied, and no aortic obstruction or mitral regurgitation, the pulse is large and powerful.
<i>Pericardial friction.</i>	Often present.	Absent.

Prognosis.—In considering the prognosis of pericarditis, we have to take into account, *firstly*, the danger to life involved by the attack itself (*i.e.* the *immediate* prognosis), and *secondly* (in those cases which recover), whether the adhesions, which almost always remain, to a greater or less extent, after a severe attack of pericarditis, will be likely to produce any permanent or after bad effect upon the action of the heart. The *remote* prognosis is practically, therefore, synonymous with the prognosis of ‘adherent’ pericardium.

The immediate prognosis.—The danger to life depends upon the following conditions :—

(1) *The ætiological cause of the condition.*—Uncomplicated cases of rheumatic pericarditis almost always recover. Peri-

carditis due to traumatic injury, wounds of the sac, etc., is less serious than pericarditis which follows the rupture of an abscess (in the liver for example) into the sac. Pericarditis occurring in the course of Bright's disease is very generally fatal; the prognosis is still worse in cases of purpura and scurvy; while tubercular is almost invariably, and cancerous pericarditis always fatal.

(2) *The severity of the associated diseased condition, and of the pericardial inflammation itself.*—Other things being equal, a *dry* is less serious than a *moist* pericarditis, while in the latter form, the more copious the effusion, the worse the prognosis. In both varieties, however, *the condition of the cardiac muscle, and the state of the valvular apparatus*, are the two most important (cardiac) elements which determine the prognosis, the presence of myocarditis being especially unfavourable.

The *form* of the pericarditis, whether acute or chronic, is also of importance, chronic pericarditis being very often fatal. When the effusion consists of pus, the prognosis is also much more unfavourable than in simple cases.

(3.) *The presence of complications, and the previous state of health of the patient.*—The opinion must of course be based upon the exact nature of the complication. Hyperpyrexia, with severe cerebral symptoms, occurring in the course of rheumatic pericarditis is a very serious complication; while the fact that the heart was diseased before the attack of pericarditis commenced, adds considerably to the danger. Other things being equal, a robust, healthy person will be more likely to survive an attack of pericarditis than a weakly delicate individual. But we find many exceptions to this general rule.

(4.) *The age of the patient.*—In very young or very old subjects, the prognosis is much more unfavourable than at other periods of life.

Treatment.—In considering the ætiology, I have pointed out that in a large proportion of cases, pericarditis is secondary; and that, in some cases, the inflammation of the pericardium occurs in the course of certain general affections

(acute rheumatism, Bright's disease, etc.); in others, it is due to the direct extension of pre-existing inflammation from some neighbouring organ or part (the pleura, mediastinum, lung, etc.); while in a third group of cases, it results from the secondary deposits of a tubercular, sarcomatous, or cancerous nature in the tissues of the pericardium. Now, in treating the many different primary affections, which are included in these three groups, it is essential to keep this tendency to pericarditis prominently in view, to protect the patient most scrupulously from everything likely to act as an exciting cause or to arouse the inflammation of the pericardial sac, in short to adopt, so far as we are able, a preventative plan of treatment.

It is no less important in managing the primary affections, to examine the heart carefully from time to time, and to look out for the first symptoms and signs of local mischief, in order that we may, if possible, cut short and allay the pericardial mischief as soon as it appears; while, in treating cases of secondary pericarditis, after the pericardial inflammation has fully developed, one of the points to which attention must be prominently directed is the treatment of the primary affection.

The prevention of pericarditis.—Speaking generally, we may say that the best method of preventing secondary pericarditis is to remove as soon as possible the primary affection, guarding the patient at the same time from everything which is likely to excite the inflammation of the pericardium itself.

Some of the primary affections, in the course of which pericarditis is apt to occur, such for instance as chronic Bright's disease and cancer, resist our present therapeutic means of cure; and in them we must content ourselves with guarding the patient against exposure to cold, and anything which will excite or depress the action of the heart. Others, as for example, acute rheumatism and some cases of local inflammation in the neighbourhood of the pericardium, can be successfully controlled by a well directed and energetic plan of treatment. In acute rheumatism, for instance, we

endeavour to cut short the attack by saturating the system with salicin or salicylate of soda; allaying excessive pain, should it be necessary, by hypodermic injections of morphia; guarding the patient from cold by placing him in blankets and a flannel night gown to absorb the excessive perspiration, by warming the stethoscope and avoiding any undue exposure of the præcordium in making a routine examination of the heart; and carefully avoiding everything which will be in the least likely to excite or injuriously depress the action of the heart.¹

It would be out of place to detail here the manner in which the different primary affections are to be dealt with, I therefore pass to the treatment of the pericardial inflammation itself.

The treatment of the pericardial inflammation itself.—In dealing with a recently developed case of pericarditis, it is essential to remember, as Dr Sturges has so ably pointed out, and as I have for a long time been in the habit of teaching, that an inflammation of the pericardium differs from many other inflammations in this important particular, viz., that although it seldom is of itself *directly* fatal, it frequently is

¹ I am aware that statistics seem to show that salicin and salicylate of soda do not prevent rheumatic pericarditis and other cardiac complications. It will not, however, be disputed, that the rheumatic symptoms proper (the fever, the joint affection and the pain) are speedily relieved by these drugs; and I find it difficult to believe that any plan of treatment, which is able to cut short the rheumatic fever itself, will not in many cases prevent the occurrence of pericarditis, provided that it is energetically enforced before the pericardial inflammation is established. Sibson's observations as to the influence of pain and cardiac excitement in the production of rheumatic pericarditis seem to lend additional support to this view. The difficulty in arriving at a conclusion from statistics as to the influence of salicin in preventing rheumatic pericarditis and other cardiac complications is considerable. In many cases, no doubt, the signs of pericarditis are first detected *after* the salicin plan of treatment has been commenced, but it is unfair to lump all cases together, and to say that salicin does not prevent rheumatic pericarditis; in some, the pericarditis was without doubt actually present though undetected or undetectable before the treatment was commenced; in others, the pericardial inflammation was developed before the system was properly saturated with the drug. It is only the remainder, viz., those in which the pericardial inflammation developed after full saturation of the system that can be legitimately adduced in evidence; and we want, I think, more information on this point. (This subject is afterwards considered in greater detail, see p. 388.)

followed by secondary changes, which cripple the vitality of the patient, and which lead to the subsequent disease, and it may be to an early death. A lung, for example, which has been affected with croupous pneumonia, however severe, is (in the great majority of instances) to all intents and purposes as sound as it was before ; but a heart after a severe attack of pericarditis is, in many cases, permanently damaged, in consequence, not only of the adhesions which remain, but also—and this is much more important—because of the strong tendency which the inflammatory process has to make its way to the deeper structures, and to leave permanent structural alterations in the muscular walls of the organ.

As soon, then, as there is any evidence of local mischief, it is all important to do what we can to cut short and allay the inflammatory process in the pericardial sac.

The patient should, if possible, be placed in a well ventilated and airy room, the temperature of which must be continuously kept between 65° and 70° Fahr. Absolute rest in the recumbent position must be rigidly enforced, and everything which tends to excite the action of the heart avoided. A full dose of quinine, in combination with morphia, as recommended by Professor Robert Bartholow,¹ should be administered ; and local means employed to restrain those vascular changes which accompany every inflammation.

In robust and previously healthy individuals, and more especially *in those in whom the pulse is strong and tense*, the local abstraction of blood should be practised, six, eight, or ten leeches being applied over the præcordial region. The continuous application of cold to the region of the heart by means of ice-bags is next recommended, more especially by German writers. Personally, I have no experience of this plan of treatment, for, in common with most English physicians, I have been in the habit of recommending warm applications or a mustard poultice, followed by the continuous application of

¹ Bartholow recommends the administration of a full dose of quinine (15-20 grains of the sulphate) with a quarter to half a grain of morphia, 'and the cinchonism should,' he says, 'be maintained by repeated smaller doses, for twenty-four hours or longer.'—*Practice of Medicine*, second edition, p. 237.

heat, by means of hot poultices or fomentations, to the præcordia. I can easily conceive, however, that in suitable cases (in robust and previously healthy individuals, and quite at the commencement of the attack) the continued application of cold may be beneficial. Excessive and violent action of the heart should be restrained by the frequent administration of small doses of aconite¹ or veratrum viride.

The administration of aconite is recommended by Professor Sydney Ringer 'in pericarditis accompanied with violent throbbings and extreme pain.'²

Pain in the region of the heart should be relieved by subcutaneous injections of morphia; and even in those cases in which there is little or no pain, the administration of small doses of opium or morphia is often very beneficial.

I must repeat, that depletion, continuous cold, aconite and veratrum viride are only permissible in robust and previously healthy individuals, especially in those in whom the pulse is strong and tense; and also, that it is important in all cases in which these remedies are employed, to keep a watchful eye upon the condition of the heart and pulse, for it is essential to avoid anything which *depresses* the action of the heart. Leeches, continuous cold, aconite and veratrum, are useful so long as they are employed merely to *moderate excessive action*, they are injurious when they are pushed beyond this point, and allowed to produce depression.

The treatment appropriate to the primary affection must, at the same time be persevered with. There is, however, one very important exception to this general rule, viz., that in rheumatic cases the further administration of the preparations of salicin (more especially salicylate of soda, for, according to Dr Maclagan, salicin itself does not depress the heart) should be dispensed with. The diet should be light and nutritious, consisting chiefly of milk and soups.

As soon as the excessive action of the heart has been moderated, or, if at the end of twenty-four or thirty-six hours

¹ In cases of albuminuric pericarditis (pericarditis occurring in the course of Bright's disease) opium if given at all, must be administered cautiously.

² *Handbook of Therapeutics*, ninth edition, p. 472.

the inflammatory process is not arrested, the aconite or veratrum should be discontinued ; and it is, in many cases, advisable to discontinue the ice-bags, and to apply poultices, fomentations, or soothing liniments, containing opium, chloroform, belladonna, etc., to the præcordium.¹

The subsequent treatment of the case must be modified in accordance with the form of the disease (whether dry or moist), the severity of the attack, the general constitutional state, etc.

It is difficult to lay down general rules applicable to all cases. The following, however, may be said to be the chief indications for treatment.

1. *To allay the inflammatory process.*—The same general measures should be continued as were recommended in the earliest stages of the attack (absolute rest, the avoidance of anything likely to excite the action of heart, a light diet, etc.). When the fever is considerable, the quinine may be continued, with or without a diaphoretic. Warm poultices or anodyne fomentations are to be continually applied to the præcordial region.

2. *To relieve symptoms.*—*Pain* is to be relieved by local anodyne applications, or the internal or subcutaneous administration of opium or morphia. *Sleeplessness*, which is often a distressing symptom, is also best relieved by an opiate at bedtime. Where there is no pain, chloral, or chloral combined with bromide of potassium, may be substituted ; personally I prefer opium or morphia, for chloral is apt to have a depressing influence upon the heart. *Nausea and*

¹ Modifications in the vascularity of the pericardium may probably be produced in two ways by means of external application, viz. :—

(1.) *Directly*, through the communications which exist between the subcutaneous vessels of the chest wall and the vessels on the outside of the pericardial sac. By depleting the subcutaneous vessels by means of leeches, we deplete at the same time the vessels on the outside of the pericardial sac ; and by producing dilatation of the subcutaneous vessels by the application of a warm poultice, we draw blood from the vessels in the exterior of the pericardium which communicate with them.

(2.) *Indirectly*, by reflex nervous impulses. This is probably the more important of the two, for by this means we can probably act not only upon the exterior of the visceral pericardium, but also upon the interior of the parietal pericardium, and indeed upon the vascularity of the heart itself.

sickness, which are not unfrequent, and are often distressing symptoms, are best relieved by sucking ice, the application of a mustard blister to the pit of the epigastrium, and the internal administration of champagne, brandy, morphia, or hydrocyanic acid. *Dyspnœa* is often met with in the later stages of the attack; it may depend, as we have previously seen, upon many different conditions, amongst which failure of the action of the heart, valvular complications, the pressure of a large pericardial effusion upon the trachea or left bronchus, and lung complications (such as pleurisy and pneumonia) are the chief. Each of these forms of dyspnœa will, of course, require a special plan of treatment. When, for instance, the shortness of breath is due to failure of the heart's action, cardiac tonics and stimulants should be freely administered; when the dyspnœa is urgent, and when it depends upon the mechanical pressure of a large effusion, aspiration of the pericardium should be practised.

Hyperpyrexia is, as we have previously seen, occasionally developed in the course of rheumatic pericarditis. Prompt measures should be at once taken to reduce the temperature, and amongst these the cold-bath treatment appears to be by far the best. The patient should be immersed in a bath at 95°–100° Fahr., cold water should gradually be added until in the course of twenty minutes or half an hour the temperature of the bath is reduced to 60° Fahr. If there is any tendency to cardiac depression, it is a good plan to give a small quantity of brandy both before and after the bath. The patient should at once be removed from the bath if any serious symptoms of cardiac depression arise. In private practice, or when it is inconvenient to immerse the patient in a bath, the application of iced-cloths, in the manner recommended by Professor Sydney Ringer,¹ may be substituted.

3. *To sustain the strength of the patient, and in particular should any indications of cardiac failure arise, to stimulate and strengthen the action of the heart.*

This is a most important indication, for one of the great

¹ *Handbook of Therapeutics*, ninth edition, p. 61.

dangers in pericarditis is failure of the action of the heart. A watchful eye should always be kept upon the pulse and venous circulation. When the heart's action is irritable and weak, digitalis or convallaria majalis should at once be administered, the dose depending, of course, upon the special circumstances of each case. Alcoholic stimulants (brandy), carbonate of ammonia, or spirits of chloroform, should be given in those cases in which there is rapid failure of the heart.

4. *To promote absorption of the inflammatory products.*—In the majority of cases, the inflammatory products are more or less rapidly absorbed soon after the acme of the effusion has been reached; but this is not always the case, and it is often desirable to assist the natural process of absorption. With this object, iodide of potassium, diuretics (especially digitalis, and convallaria majalis), and if the patient's strength permits, diaphoretics are administered internally; counter irritants (tincture of iodine, small and frequently repeated fly-blisters, etc.) are applied over the præcordia; while the greatest attention is paid to the condition of the general health, the treatment in fact should, so far as possible, be of a tonic character, the object being to raise the general tone of the system, and in particular to sustain and strengthen the action of the heart. Digitalis and convallaria majalis, which are both powerful cardiac tonics and active diuretics, are particularly useful. The diet should be light, and at the same time as nutritious as possible; the bowels must be regulated; and the ventilation of the patient's room carefully attended to. It is particularly important to avoid all causes of mental depression, and in short to keep the patient as cheerful and hopeful as possible.

5. *When the effusion is progressing so rapidly or is so extensive as to seriously embarrass the action of the heart and endanger life, or when it refuses to be absorbed by ordinary treatment, to withdraw the fluid by aspirating the pericardial sac.*

It is unnecessary again to detail the symptoms, which result from the pressure of a large pericardial effusion upon the heart itself and upon the surrounding structures, or the

manner in which a pericardial effusion is to be distinguished from the other conditions with which it is likely to be confounded, for these points have already been considered (see pages 329, *et seq.*). I therefore pass on to say that the operation has now been performed in a large number of cases with complete success ; the result has been most favourable in rheumatic cases associated with rapid effusion into the sac ; it is much less favourable in non-rheumatic forms, and especially when the condition has become chronic. Dr John B. Roberts of Philadelphia, who has directed special attention to the subject, states that of thirty-five cases in which the operation has been performed since the year 1860, ten recovered and twenty-five died, giving a mortality of 71·42 per cent. 'In the twenty-five instances where death occurred subsequent to the tapping serious disease is stated to have existed in all the cases except three. In other words, out of the whole thirty-five cases operated upon, there were thirteen cases of pericardial effusion where other diseases did not seem to act as a complication, and of these ten recovered and three died. This would give a mortality of 23 per cent.'

Dr Roberts not only recommends that the operation should be performed 'whenever the effusion whether it be serum, pus, or blood accumulates so rapidly or in such quantity that it threatens to destroy life, and refuses to undergo absorption by ordinary treatment ;' but he even advocates its use as a palliative method of relief, in cases in which the pericardial inflammation depends upon, or is associated with incurable organic conditions.

The sac should be punctured with the aspirator, and with the strictest antiseptic precautions. In selecting the point of puncture we must avoid the heart itself, the diaphragm, the internal mammary artery, and, if possible (though this is a point of much less importance) the left pleura. In most cases the needle should be inserted in the fifth left interspace, midway between the nipple line and the sternum, just above the junction of the sixth rib with its

¹ These statistics, which are detailed in Dr Robert's work on *Paracentesis of the Pericardium*, page 97, were brought up to the year 1880.

costal cartilage. When the heart is enlarged or tied down by adhesions, the puncture should be made in the sixth interspace, or, as Sibson suggests, between the ensiform cartilage and the seventh costal cartilage. The following are the directions which it is advisable to follow in performing the operation:—

(1.) Place the patient in the recumbent position, so as to allow the heart to fall away from the front wall of the chest.

(2.) See that the apparatus is in working order and perfectly clean. It is a good plan to draw a stream of carbolic solution through it before attempting to insert it into the sac.

(3.) Count the interspaces accurately from above, downwards.

(4.) Percuss and auscultate over the seat of the proposed puncture, and satisfy yourself that it is dull on percussion, and that the dulness does not depend upon the presence of the heart.

(5.) Introduce the needle of the aspirator with a firm plunge; if the skin is thick, or if you are using a large needle, a preliminary incision through the skin with a sharp bistuory is advisable. The needle should be directed at first directly backwards, and then backwards and downwards in order to avoid wounding the heart.

(6.) If after exhausting the apparatus no fluid passes, withdraw the needle, wash it out, and if you feel sure of your diagnosis reintroduce it.

(7.) It is advisable, unless there is any indication to the contrary, to draw off all or a large portion of the fluid at one sitting.

(8.) After the needle is withdrawn a piece of lint should be fixed by adhesive plaster over the superficial wound.

(9.) If pure blood flows through the instrument, if the heart has been wounded, or if any untoward symptoms, such as acute pain, dyspnœa, etc., should arise during the process of aspiration, the needle should at once be withdrawn.

6. When the effusion consists of pus to lay open the sac, under strict antiseptic precautions, and to insert a drainage tube.

When the effusion consists of pus the case should, in short, be treated as any other internal abscess. Both physicians and surgeons are becoming every day more and more convinced that whenever there is an internal collection of pus it should if possible be evacuated; and a collection of pus within the sac of the pericardium forms no exception to the general rule. As yet the operation has only been twice performed successfully. The details of the last case, that of Dr West, will be found in the *Lancet* for April 28, 1883, p. 728, to which the reader is referred for further information on the subject.

ADHERENT PERICARDIUM.

The recovery from an attack of acute pericarditis is seldom complete, for in most cases some adhesions between the visceral and parietal layers of the sac remain. Sometimes the whole sac of the pericardium is obliterated, more frequently the adhesions are partial.

The adhesions are permanent,¹ and since they are in some cases followed by secondary changes in the heart and circulation, and therefore by permanent bad effects upon the health of the patient, I must now describe the nature of the secondary changes, the symptoms and physical signs to which they give rise, and the manner in which pericardial adhesions are to be recognised during life.

The effects which pericardial adhesions produce upon the heart, and the symptoms to which they give rise, depend more particularly upon the following conditions:—

1. The extent and character (more especially the density) of the adhesions.
2. The condition of the muscular walls of the heart after the attack of pericarditis has subsided.
3. The condition of the valvular apparatus of the heart.

When the adhesions are limited in extent, when the myocardium and valvular apparatus remain healthy, the adhesions produce little or no effect upon the condition of the heart, and are unattended with symptoms.

¹ It occasionally happens that the adhesions are afterwards broken down by the movements of the heart.

When, on the contrary, the adhesions are universal and dense, when the pericarditis is complicated with myocarditis, and an increase of the intermuscular fibrous tissue remains after the attack has subsided, the muscular walls of the heart usually undergo degeneration, the muscular fibres becoming atrophied¹ in consequence of the pressure which the fibrous tissue (both on the surface and in the substance of the organ) exerts upon them during its cicatrisation.² In these cases very grave symptoms, indicative of failure of the heart muscle, which I shall describe in detail when I come to speak of the effects of myocârditis (see p. 564), are observed.¹

In other cases, again, the heart becomes hypertrophied ; indeed it used to be supposed that an adherent pericardium, by embarrassing the action of the organ, necessitated increased force of contraction, and consequently produced hypertrophy. We now know that although this is the sequence of events in some cases, partial atrophy of the muscular fibres and dilatation rather than hypertrophy are usually observed. In many cases a combined condition of partial atrophy with dilatation, and of partial hypertrophy results ; and in a certain number of cases more or less general hypertrophy is found. This result (hypertrophy) is usually due to associated valvular lesions, though it may be the direct result of the pericardial adhesions independently of any valvular lesion ; it is most likely to occur in those cases in which the pericardial sac is adherent to the front wall of the chest as well as to the surface of the heart, and in which the myocardium is healthy.³

In cases of this description (*i.e.* adherent pericardium with

¹ In many of these cases the weight of the heart and the thickness of its walls are not diminished, in fact they may be increased. The atrophy of the muscular fibres can, in such cases only be detected by microscopical examination.

² The fibrous tissue which remains after a pericarditis, contracts just as the fibrous tissue in a cicatrix does

³ It must be remembered that increased thickness of the cardiac walls, and increased weight of the organ, do not necessarily indicate *true* hypertrophy, *i.e.* an actual increase of the muscular tissue. The increased weight may be due to an increase of fibrous tissue and fat, and may actually be associated with atrophy of the true muscular elements, as I have observed above.

hypertrophy) the symptoms are usually those of the valvular lesion which is so often present, and on which, to a considerable extent at least, the hypertrophy depends.

• *The physical signs of adherent pericardium.*—In some cases, more especially when the adhesions are partial and confined to the interior of the sac, the condition is not attended with any distinct physical signs, and cannot, therefore, be recognised during life; in other cases, there are very distinct physical signs which clearly indicate the nature of the condition; while in a third group of cases, the physical signs are equivocal or indistinct.

The physical signs characteristic of adherent pericardium are best marked in those cases in which—

(1) The adhesions within the sac are extensive or universal.

(2) The exterior of the sac is adherent to the chest wall and to the anterior margins of the lungs.

(3) The heart is acting powerfully, *i.e.* is hypertrophied.

The condition of the anterior margin of the lungs, more especially of the left lung, is also an important point. When the anterior margins of the lungs are tied down by adhesions, the physical signs are more distinct. When, on the contrary, the lungs have free play, and still more when they are voluminous or emphysematous, the physical signs of adherent pericardium are obscure and indistinct.

When there are extensive adhesions both on the interior and exterior of the pericardial sac, the heart is, as it were, rigidly fixed in the cavity of the chest, and the free play of its natural movements is interfered with. In consequence of this alteration in its physical condition, the following alterations in the physical signs are observed:—

(1) *Systolic depression, instead of systolic protrusion of the apex beat; and in many cases a systolic depression of the lower end of the sternum and adjacent costal cartilages.*—In consequence of the adhesions, the ventricles, during their contraction, pull directly upon the diaphragm and front wall of the chest, and it is to this traction, aided, of course, by the

external pressure of the atmosphere, that the systolic depression of the apex beat and front wall of the chest is due. This systolic depression is most marked when the chest wall is yielding (*i.e.* in young subjects), and when the ventricles are acting powerfully (*i.e.* when the heart is hypertrophied); *vice versâ* it is absent or indistinct when the cartilages are ossified, and when the heart is acting feebly (*i.e.* the muscular walls of the heart degenerated, its cavities dilated, etc.)

Systolic depression, instead of depression of the apex beat, is not absolutely distinctive of pericardial adhesions, as Friedreich has shown; and Bauer says—‘We may state in general terms that systolic pitting at the site of the apex beat may be present in all cases in which the normal movement of the heart downwards and to the left, with elevation of the apex, is hindered, provided at the same time the lungs do not sufficiently approach each other, and the contraction of the heart is powerful enough to force the apex away from the chest wall. Under such circumstances the pitting follows, as a matter of course, from atmospheric pressure.’¹

(2) *Permanent depression of the præcordial region.*—This condition, in which, as it were, the temporary systolic depression has become fixed and permanent, is only observed in those cases in which the chest wall is elastic, and in which the pericardium is extensively adherent to the heart on the one side and to the chest wall on the other.

(3) *A diastolic rebound of the chest wall.*—This is a most distinctive sign of adherent pericardium, and it is due to the fact that after the depression of the lower end of the sternum and adjacent costal cartilages has taken place, and when the ventricle begins to relax, the natural elasticity of the chest wall causes a rebound, which can be distinctly felt by the hand as an impulse during the diastole of the heart.

(4) *Absence of the apex beat.*—In some cases of adherent pericardium the apex beat is absent. This condition is most likely to occur when the heart is acting feebly, or when the lungs are adherent over the position of the apex beat.

In some cases in which systolic depression of the apex beat is observed, it is impossible to feel the pulsation of the

¹ Ziemssen's *Cyclopædia of Medicine*, vol. vi. p. 643.

apex impulse; in other cases a diastolic impulse, similar in character to the diastolic rebound of the chest wall, has been noted.

(5) *Fixation of the apex beat.*—In conditions of health certain alterations in the position of the patient are attended with corresponding alterations in the position of the apex beat. Sibson, for example, has shown that when the patient lies on his left side, the apex beat may be displaced ‘from the fifth interspace, a little lower than the nipple, and within the mammary line, to the sixth or seventh space, two inches to the left of the line.’ Now, in the conditions which we are considering, this—the natural mobility of the apex beat—cannot, of course, take place; and whatever the position of the patient the apex beat remains much in the same position.

(6) When the lungs are at the same time retracted and fixed by adhesions, *the area of pericardial dulness is not only enlarged, but remains of the same size both during inspiration and expiration.*

This is not, of course, distinctive of pericarditis, for it only shows that the anterior margins of the lungs are tied down as the result of pleurisy; but when taken in conjunction with the other physical signs, mentioned above, it is corroborative of the presence of an adherent pericardium.

(7) *Distention of the veins of the neck during the systolic depression of the chest wall* (i.e. during the systole of the ventricles), followed by their rapid collapse and disappearance during the diastolic rebound (i.e. during the diastole of the heart) is another important sign, which was first described by Friedreich. It is due to the fact, that during the systolic depression of the chest wall, the cavity of the chest is diminished in size, and a mechanical hindrance offered to the return current of blood through the superior cava. During the diastolic rebound, on the other hand, the cavity of the chest is suddenly expanded, a suction force is exerted, and the previously distended veins are in consequence suddenly emptied.

Indurative-mediastino-pericarditis. When in addition to the adhesions which rigidly fix the heart, as it were, in the cavity of the thorax, the cellular tissue of the mediastinum is implicated by the inflammation, and fibrous adhesions are formed between the chest wall and the lungs on the one hand, and the great vessels at the base of the heart¹ (the aorta and its branches and the superior vena cava) on the other, the passage of the blood from the heart through the aorta and its branches, and to the heart through the superior cava, may be interfered with.

This interference with the circulation, through the aorta and vena cava, only occurs when the adhesions are put upon the stretch; and it is due to the fact that the *tense adhesions* constrict the blood vessels, and thereby produce a mechanical hindrance to the circulation. And since the adhesions are put upon the stretch by the expansion of chest wall and lungs which occurs during the effort of inspiration, it follows that the interference with the circulation through the aorta and its branches, and through the vena cava superior is only observed during the same period, *i.e.* during inspiration.

The obstruction to the passage of the blood through the aorta produces a peculiar alteration in the character of the pulse, to which the terms *pulsus paradoxus* and *pulsus inspiratione intermittens* have been given. As the latter term indicates, the alteration consists in the fact that the pulse becomes much diminished in volume, or altogether effaced during the act of inspiration. (See figs. 161, 162, 163.) It is said to be always more or less quickened, small and intermittent;

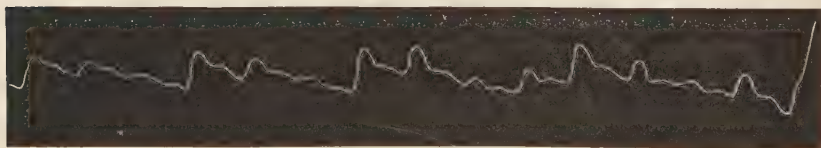


FIG. 161.—*The pulsus paradoxus.* (After Kussmaul.) Copied from Ziemssen's *Cyclopaedia*, vol. vi. p. 650.

¹ The term *indurative-mediastino-pericarditis* has been given to cases of this description.

and it may be irregular, while the action of the heart is at the same time perfectly rhythmical and regular.¹

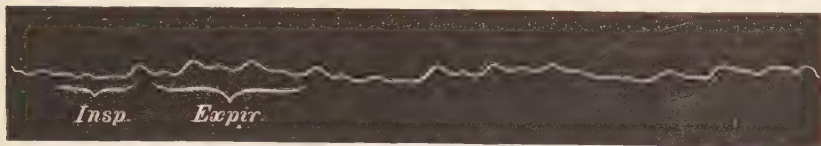


FIG. 162.—*The pulsus paradoxus.* (After Ziemssen) *Cyclopædia of Medicine* vol. vi. p. 656.



FIG. 163.—*The pulsus paradoxus.* (After Traube.) Copied from Ziemssen's *Cyclopædia*, vol. vi. p. 654.

The obstruction to the passage of blood through the superior cava causes distention of the jugular veins ; and since the obstruction only occurs during inspiration, the jugular veins become distended instead of collapsing during inspiration (as they do in health), the distention rapidly subsiding during expiration, that is, when the constrictive bands are relaxed, and the obstruction to the blood in the superior cava is removed.

Diagnosis.—To sum up, then, it is impossible in some cases to diagnose an adherent pericardium ; in others, the diagnosis is difficult ; while in some, the condition can be easily and surely recognised.

A history of pericarditis is presumptive evidence of the condition, for some adhesions usually remain after inflammation of the pericardial sac. Systolic depression, instead of systolic protrusion, of the apex beat, is strongly suggestive of

¹ The pulsus paradoxus is not pathognomic of 'indurative-mediastino-pericarditis,' though it is strongly suggestive of that condition—it has been observed in ordinary pericarditis without external adhesions ; also in some cases in which there was an obstruction to the entrance of air into the thorax (stenosis of the air passages). I myself have seen a very similar pulse during an attack of spasmodic asthma. (See fig. 103.)

an adherent pericardium ; and the supposition is strengthened by an immoveable apex, and an increased area of cardiac dulness with fixation of the anterior margins of the lungs, more especially of the left. The diagnosis is confirmed by a permanent depression over the præcordial region.

Systolic depression of the lower end of the sternum and adjacent costal cartilages, with a diastolic rebound of the chest wall, and distention of the veins of the neck during the systolic depression of the chest wall, followed by their rapid collapse and disappearance during the diastolic rebound, are the strongest possible evidence of the condition.

Indurative-mediastino-pericarditis may be strongly suspected when the pulse presents the *paradoxical* character ; and may be positively diagnosed when, in addition, there is distention of the cervical veins during inspiration, followed by their rapid collapse and disappearance during expiration.

Prognosis.—*Per se*, an adherent pericardium is of no practical importance ; but since it often leads to secondary changes in the condition of the heart, the prognosis should always be guarded, and the case carefully watched. The prognosis entirely depends, therefore, upon the effects which the adherent pericardium is likely to have, or has already had, on the muscular walls and cavities of the heart, and upon the presence or absence of valvular complications.

The prognosis is most favourable in those cases in which the cardiac muscle is sound, in which there are no valvular lesions, and in which the adhesions are not extensive.

Extensive adhesions—evidenced during life by marked systolic depression of the lower end of the sternum, followed by a notable diastolic rebound—lead in most cases, sooner or later, to serious structural alterations in the heart. When valvular lesions complicate the case, the prognosis is as a rule bad ; but there are many exceptions to this general rule, depending upon the nature and extent of the valvular lesion, etc. When the myocardium is implicated, the prognosis is most unfavourable.

Treatment.—Nothing can be done in the way of direct treatment, *i.e.* removing the adhesions. The chief indications are to maintain the general health in the highest possible state of efficiency, and in particular to preserve the nutrition and strength of the heart; to avoid all strain and over exertion; to treat the valvular complications, and to relieve symptoms. The manner in which these indications are to be carried out will be more appropriately considered when I come to treat of myocarditis and valvular lesions.

CHRONIC PERICARDITIS.¹

Ætiology and Pathology.—Chronic inflammation of the pericardium is much less frequent than the acute form of the affection. In some cases, the inflammation commences slowly and gradually, in other words, it is chronic from the first; in others, an inflammation which was at first acute, does not resolve, but passes on to the chronic condition. In both instances there is usually some cause of general constitutional depression present; rheumatic pericarditis in a previously healthy person very rarely assumes the chronic form; the tubercular and cancerous forms of pericarditis, on the contrary, are, as a rule, subacute or chronic. The effusion in cases of chronic pericarditis is usually considerable, and often very great; in many cases it is hæmorrhagic or purulent in character.

Symptoms and physical signs.—After the full description which has been previously given, of the symptoms and signs of acute pericarditis, it is unnecessary to say much with regard to the chronic form. The constitutional depression is, as a rule, more considerable than in the acute variety, symptoms and signs of depression and failure of the heart's action are, in fact, amongst the most prominent features of the case. When the effusion is considerable, and when, as very often happens, the myocardium is extensively affected by the inflammatory process, symptoms due to mechanical derangement of the circulation and to the pressure of the distended pericardial sac upon the adjacent organs and parts, are usually present.

¹ I do not apply the term chronic pericarditis, as some writers do, to cases of adherent pericardium.

In some cases, the temperature is not altered, but as a rule there is some increase, more especially of the evening temperature. When the effusion consists of pus, ups and downs of temperature with rigors and sweatings may occur, but these alterations are perhaps more common during the period of pus-formation than in the after stages of the case, *i.e.* when the condition has become chronic.

The physical signs are the same as those which have been described in treating of acute pericarditis with effusion.

Diagnosis.—The diagnosis is to be arrived at in the same manner as has been described in treating of the diagnosis of acute pericarditis. The differential distinction of chronic pericardial effusion, of hydropericardium, and of enlargement of the heart itself (more especially chronic pericarditis and cardiac dilatation), is often extremely difficult, but can usually be made by careful attention to the points detailed in tables V., VI., VII. (See pages 332, 333, 334.)

Prognosis.—The prognosis of chronic pericarditis is usually unfavourable, each case must, however, be judged on its own merits. The severity of the symptoms, the amount and character of the effusion, the exact nature of the pathological condition (whether the pericarditis is rheumatic, tubercular, cancerous, etc.), and the condition of the other systems and organs (*i.e.* the presence or absence of complications), are the chief points which should be taken into consideration.

Treatment.—The object of treatment is to promote absorption of the inflammatory products, and to restore the pericardium to its natural condition. The means by which this end is to be attained, have already been described. (See the *headings* 4, 5, and 6, treatment of acute pericarditis, pages 342 to 345.)

HYDROPERICARDIUM.

Definition.—The presence of simple non-inflammatory fluid in the pericardial sac.

Ætiology.—A certain amount, half an ounce to two ounces, of clear serous fluid is very commonly found in the sac of the pericardium after death, and is probably poured out during the

act of dying, or immediately after death. It is only when this amount is considerable, and when the fluid is present before the act of dying is established, that the condition can properly be termed hydropericardium. Dropsy of the pericardium is almost always part and parcel of a general dropsy, and is usually a late event in the course of cardiac or renal disease; but while this is the clinical and pathological fact, it cannot of course be denied that hydropericardium may be local, and that it may result from an obstruction to the return current of blood from the walls of the heart and tissues of the pericardium. I have myself seen more than one case in which the amount of fluid in the pericardium was quite out of proportion to the amount of general dropsy, and in which I have little doubt that there was some local cause for the condition, such as obstruction of the coronary veins. Unfortunately the cases, which occurred some years ago, were not examined with the attention that they deserved, and I must content myself with recording the bare fact of their occurrence.

Symptoms.—It is seldom that the amount of fluid is sufficiently great to seriously interfere with the action of the heart. The symptoms are, therefore, in most cases those of the primary condition—cardiac or renal disease with dropsy. When the pericardial dropsy is considerable, effusion into the pleural sacs, and other pulmonary complications are, as a rule, present. In many cases there are also other cardiac complications, such as valvular disease, dilatation, fatty degeneration, etc. The physician must hesitate, therefore, in ascribing the dyspnœa and other symptoms of embarrassed cardiac action, which are generally prominent in cases of this description, to the pericardial effusion; these symptoms are usually for the most part due to other conditions, though the embarrassment of the heart will of course be increased when the pericardial dropsy is great, and more especially when a large effusion is rapidly poured out.

Physical signs and Diagnosis.—The presence of simple non-inflammatory fluid in the sac of the pericardium is of course attended by an increase of the præcordial dulness;

the outline of the dulness corresponds to that which has been described, in speaking of inflammatory effusion. The dulness due to simple dropsy of the pericardium is often difficult to detect, for, in many cases it is inconsiderable in amount, and in most cases the heart is at the same time enlarged. Under such circumstances it is very difficult or impossible to distinguish the dulness derived from the enlarged heart from the dulness due to the pericardial fluid. When the pericardial effusion is considerable, the cardiac impulse and cardiac sounds may be feeble or indistinct, but the character of the impulse and of the sounds depends for the most part upon the condition of the heart in other respects. All that can with certainty be said is, that a considerable amount of effusion will interfere with the conduction of sound, and that the cardiac sounds (whatever their character, whether normal or abnormal) will probably be weaker than they were before the occurrence of the effusion. In other respects they will probably be unmodified. Pericardial friction is not of course present; this fact, and the absence of pyrexia (unless some inflammatory complication should happen to be present), together with the circumstance that the pericardial effusion is, as a rule, a late event in the course of general dropsy, are the chief points on which reliance must be placed in distinguishing simple non-inflammatory fluid in the pericardium from chronic pericarditis. I need not go into further details, but must refer the reader to table V., p. 332. I must, however, repeat, that the diagnosis of simple pericardial effusion is in most cases difficult and sometimes impossible.

Prognosis.—The prognosis must be entirely guided by the primary cause of the condition; it must, of course, be remembered that dropsy of the pericardium, sufficient to be recognised during life, seldom occurs except in the later stages of general dropsy, and that in cases of this description (*i.e.* cases in which it does occur in sufficient quantity to be recognised) the prognosis is, as a rule, most unfavourable. An exception occurs in some cases of acute Bright's disease, in which condition the prognosis is much more favourable.

Treatment.—The treatment must, for the most part, be

directed to the primary condition (the cardiac or renal disease) on which the pericardial effusion depends. The general treatment is in fact that of an advanced case of cardiac or renal dropsy. Digitalis, purgatives, etc., in those cases in which there is evidence of localised venous engorgement and over-distention of the right heart, local or even general venesection, are the most useful measures which can be employed. Counter irritation (iodine, blisters) over the pericardium is sometimes useful. When the effusion is very great, and more especially when it is poured out with sufficient rapidity to seriously embarrass the action of the heart, paracentesis pericardii is advisable; no case of this description has come under my own observation.

PNEUMOPERICARDIUM.

Definition.—The presence of gas in the bag of the pericardium.

Ætiology and Pathology.—Pneumopericardium is extremely rare. The gas almost invariably enters the pericardium from without, occasionally as the result of wounds of the chest wall (stabs, the operation of paracentesis pericardii) more frequently in consequence of a communication being established, as the result of disease, between the sac of the pericardium and some of the surrounding air-containing viscera (the œsophagus, lungs, stomach). It has also been supposed that gas may be secreted by the lining membrane of the pericardium, or, may be produced as the result of the decomposition of inflammatory (purulent) fluid within the sac. It is not at all uncommon in advanced states of decomposition to meet with fœtid gas in the sac of the pericardium after death, but these cases, which must of course be carefully distinguished from true pneumopericardium, do not support the occurrence of decomposition during life. Pneumopericardium is (?) always associated with pericarditis, and since the inflammatory products are almost always purulent, pneumopericardium is practically synonymous with the condition to which the term *pyo-pneumopericardium* has been applied.

Symptoms and physical signs.—The symptoms are those of pericarditis, usually of purulent pericarditis. In many

cases symptoms and signs of disease in the surrounding viscera (cancer of the œsophagus, abscess in the liver, disease of the lung, etc.) are also present. The physical signs are characteristic of the presence of gas and fluid. The percussion note is tympanitic over the position of the gas; and it is needless to say that this varies with the position of the patient, since, when gas and fluid are both present, the fluid necessarily gravitates to the lower level. On auscultation, splashing, gurgling, churning sounds, often of a metallic pitch, and sometimes auto-audible, which correspond in rhythm to the cardiac contractions, are heard.

Diagnosis.—The diagnosis does not, as a rule, present any difficulties; the condition must, of course, be distinguished from others in which a cavity containing air and fluid is in close contact with the heart (pneumo-thorax, a cavity in the lung, and a distended stomach), the position of the resonance (over the centre of the heart when the patient lies on his back), the fact that the splashing sounds are synchronous with the cardiac contractions, the nature of the symptoms, and the character of the associated physical signs, could hardly fail to enable a careful observer to come to a correct conclusion as to the nature of the case.

Prognosis.—The prognosis is very unfavourable, though not so hopeless as might *à priori* be supposed. The nature of the primary affection is one of the most important elements to be taken into consideration in attempting to determine the future course of the case. When the condition is due to traumatic injury, the prognosis is more hopeful than in any other form; when, on the contrary, the condition results, as it did in the case related by Begbie,¹ from the perforation of a cancerous tumour of the œsophagus, the prognosis is of course hopeless. Each case must therefore be judged on its own merits, special attention being given to the ætiology and the points which guide the physician in forming a prognosis in purulent pericarditis.

Treatment.—The same treatment which has been recommended for purulent pericarditis must be carried out. With

¹ *Edinburgh Medical Journal*, 1862.

rare exceptions, such, for instance, as pneumopericardium the result of cancerous perforation, the sac of the pericardium should be laid open and a drainage tube inserted, the strictest antiseptic precautions being of course observed, during the operation and the subsequent progress of the case.

HÆMOPERICARDIUM.

When pure blood is found in the cavity of the pericardium after death, it has usually escaped either from a rupture in the heart itself, from an aortic aneurism, or from a rupture of the coronary artery. The symptoms and physical signs which characterise cases of this description are detailed in other parts of this work. (See spontaneous rupture of the heart, aneurism of the thoracic aorta.)

Those cases of pericarditis in which the inflammatory, exudation is bloody, have been already referred to. (See page 301.)

CHAPTER V.

THE DISEASES OF THE ENDOCARDIUM. ACUTE SIMPLE ENDOCARDITIS. ULKERATIVE ENDOCARDITIS. CHRONIC ENDOCARDITIS. CHRONIC VALVULAR LESIONS. MITRAL INCOMPETENCE. MITRAL STENOSIS. AORTIC INCOMPETENCE. AORTIC STENOSIS. TRICUSPID INCOMPETENCE. TRICUSPID STENOSIS. PULMONARY STENOSIS. PULMONARY INCOMPETENCE.

THE endocardium, or membrane which lines the interior of the heart, and which, at the auriculo-ventricular and arterial orifices is folded on itself so as to form the valve segments (auriculo-ventricular and sigmoid¹ valves) is liable to be affected by acute inflammation and by chronic inflammatory and degenerative processes. All of these conditions are of great practical importance, more particularly from the fact that they are the most common and important causes of cardiac valvular lesions.

In studying the diseases of the endocardium the following facts should be remembered:—

1. That these affections are seldom primary.
2. That the valve segments and chordæ tendineæ are the parts of the endocardium on which the stress of the lesion, so to speak, falls.²

¹ The endocardial or inner lining membrane of the heart is prolonged over the sigmoid (aortic and pulmonary) valves, and becomes continuous with the inner coat of the artery.

² The valve segments are often the only parts of the endocardium (to the naked eye at least), which appear diseased, and in almost all cases they are the parts which are first and most affected. The greater tendency which diseased processes have to locate themselves on the valves, is probably due to the fact that the valvular segments are the parts of the endocardium which are most exposed to mechanical irritation and shock, but to this point I will again refer in speaking of the tendency of particular parts of the valve to be affected by acute inflammation.

3. That after birth the affections of the endocardium are usually left sided.¹

Let us now consider the affections of the endocardium in detail.

ACUTE ENDOCARDITIS.

Definition.—Acute inflammation of the endocardium.

Varieties.—Several different varieties of acute endocarditis have been described, but for practical purposes two chief pathological forms are, I think, sufficient, viz. :—

1. Acute simple endocarditis.
2. Acute ulcerative endocarditis.²

ACUTE SIMPLE ENDOCARDITIS.

Ætiology.—The causes of acute simple endocarditis are very similar to the causes of acute pericarditis.

The condition is almost always secondary, a large proportion of the cases being *rheumatic* (occurring more particularly in the course of acute and subacute articular rheumatism). Scarlet fever is another affection, which, as Dr Sansom more particularly has insisted upon, is often associated with or followed by endocarditis. Inflammation of the endocardium is met with in a considerable number of cases of chorea,³ it also occasionally occurs in connection with measles, puerperal fever, pyæmia, diphtheria, typhoid, etc.

¹ Various theories have been put forward to account for the fact that the left endocardium is so much more frequently affected by disease than the right. It has been supposed, for example, that the different character of the blood in the two hearts was the cause of the condition; the left heart, which contains arterial blood, being, it is theorised, more liable to inflammatory changes than the right heart, which contains venous blood. A more probable explanation is that which accounts for the condition by the fact, that the valves of the left heart are more exposed to irritation and shock, in consequence of their being more forcibly and violently closed, than those of the right.

² It is a disputed point whether there is any essential and primary difference between these two forms. Some authorities say that it is the constitutional condition, of the individual who is affected by the endocardial inflammation, which determines the form of the lesion (whether simple or ulcerative) rather than any essential difference in the nature of the inflammatory process.

³ In many cases of chorea the endocarditis is also rheumatic.

Authorities differ as to the exact frequency with which endocarditis occurs in rheumatic fever. Most English and French observers place the proportion as high as one in two or three, while many German physicians make it as low as one in five or six.¹ The discrepancy is probably due to the fact that observers are not agreed as to the clinical indications of endocarditis during life.

Opinions also differ as to the influence which the severity of the attack of rheumatic fever has in producing the endocardial mischief. Some authorities, Sibson² for example, state that the more severe the rheumatic attack, the greater the tendency to endocardial inflammation. Others, such as Sansom, have not observed any such relationship; Sansom, indeed, believes 'that in the child endocarditis can arise and progress without special symptoms, without pyrexia, without the disturbing influence of any acute disease,'³ and he states that this form of endocarditis presents 'no obvious difference from the essential features of rheumatic endocarditis, such as we find in the undoubtedly rheumatic subjects.'⁴

Inflammation of the endocardium should not be looked upon as a complication but as a component part of articular rheumatism; it may occur at any period of an attack, and may even precede the joint affection. In many cases the two conditions (the joint affection and the endocarditis) are probably developed simultaneously. In the cases observed by Sibson, a systolic mitral murmur, which he considered a certain sign of endocarditis, was present in one-fourth before the end of the first week, and in two-thirds before the end of the second week; and it is important in this connection to remember that the endocardial inflammation must have been going on for some time before the systolic murmur was observed, for considerable changes must be produced in the

¹ Sibson states that endocarditis, either alone or in the form of endo-pericarditis, was present in half of the 325 cases of acute rheumatism which were under his care in St Mary's Hospital during the years 1851 to 1866, and that in one-half of the remainder the occurrence of endocarditis was either threatened or probable.—*Russell Reynolds' System of Medicine*, vol. iv. p. 526.

² *Russell Reynolds*, vol. iv. p. 199.

³ *Lettsomian Lectures*, p. 23.

⁴ *Loc. Cit.*, p. 23.

valve segments or myocardium before incompetence is established;¹ in other words, the systolic apex murmur indicative of mitral regurgitation, and which is the chief clinical sign of endocarditis, is not developed immediately the valvular inflammation is established.

Amongst the predisposing causes, previous attacks of endocarditis, the presence of structural alterations in the valves, the result of previous endocarditis and of chronic degenerative changes, and any alterations (such as congenital malformations) which expose the valves to a greater amount of mechanical irritation and shock than they are normally subjected to, must be mentioned.

After puberty the two sexes are equally predisposed to acute endocarditis.² Children seem more liable to be affected than adults.³ Anything which debilitates the system as a whole (such as poverty, exposure, etc.), more especially anything which predisposes to acute articular rheumatism, predisposes to the condition. A hereditary tendency to rheumatism must therefore be mentioned as a predisposing cause.

Pathology and Morbid Anatomy.—Acute simple endocarditis is almost always secondary. It is, in the majority of cases, so far as we can judge by the naked eye, limited to the valvular apparatus of the left heart, though acute

¹ Sibson indeed goes so far as to say, 'we are therefore, I conceive, warranted in assuming that in a considerable number of cases, the active stage of the endocarditis is passing away at the time of the appearance of the murmur.'—*Russell Reynolds' System of Medicine*, vol. iv. p. 475.

² The preliminary report of the Collective Investigation Committee of the British Medical Association seems to show that 'between the ages of eleven and fifteen, acute rheumatism is three times more frequent among females than males; between sixteen and twenty the numbers are practically equal; while, after this age, the disease is far more frequent among males than females up to the age of forty-five, when they again become equal.'—*The Collective Investigation Record*, p. 121. The fact that young females suffer so much more frequently from acute rheumatism than young males, is probably the reason why they are so much more frequently affected with mitral stenosis. Possibly, too, as Dr Barlow has suggested, the greater liability of female children to chorea, in which endocarditis is of common occurrence, is another reason why mitral stenosis occurs more frequently in women.

³ Rosenstein differs from most authorities on this point, for he considers 'the disposition to endocardial affections, on the whole, smaller in childhood than after puberty.'—*Ziemssen's Cyclopædia of Medicine*, vol. vi. p. 85.

endocarditis of the valvular apparatus of the right heart, more especially of the tricuspid valve, is, I believe, more common than is generally supposed. (Like the acute endocarditis of chorea, right-sided endocarditis is, I think, in many cases completely curable.) The mitral valve is more frequently affected than the aortic, a fact which Sibson states is due to the circumstances 'that the mitral flaps press against each other when the valve is shut, with much greater tension, force, and concentration, than the margin of contact of the aortic valve, under the triple agency of a finer margin of contact, greater pressure of blood and the muscular force and tendinous traction proper to the valve.'¹ The chordæ tendineæ are frequently implicated. In a few cases the endocardium, lining the cavities of the left auricle and left ventricle is involved. It is quite exceptional to find the inflammation limited to the right heart.²

In the earliest stages of the process, which we comparatively seldom have an opportunity of observing after death, little nodulated swellings of a rosy tint and jelly-like consistence are seen at the margins of the affected segments, more especially on the auricular surface of the mitral and on the ventricular surface of the aortic valve around the corpora Arantii. Should the endocardium lining the cavities of the heart be involved, the affected portion of the membrane seems to have lost its polish and to be somewhat opaque; and distended blood vessels can sometimes be observed coursing over, or more correctly *under*, the affected portion of the membrane.³

A little later the vegetations, which are the most characteristic and striking morbid appearances of acute endocarditis, are observed. Now, since the inflammatory changes are always first and most marked on those portions of the valves,

¹ *Russell Reynolds' System of Medicine*, vol. iv. p. 458.

² This statement does not of course apply to the fœtus and newly born child, in whom the right endocardium is much more frequently involved than the left.

³ Capillary blood-vessels are 'numerous in the pericardial and endocardial membrane, including the valves' (Klein and Noble Smith, *Atlas of Histology*, page 148); and vessels of some size are met with here and there just beneath the endocardium, *i.e.* between the endocardium and myocardium in the sub-endocardial connective tissue.

which are most exposed to friction and pressure, the vegetations are generally first seen on the auricular surface of the mitral and on the ventricular surface of the aortic segments, not, it must be noted, on the free edges of the valve, but on the *lines of maximum contact*, which, as Sibson has shown, are not the free margins, but those portions of the auricular surfaces of the mitral and of the ventricular surfaces of the aortic segments which are situated just within the free margins.¹ (See figs. 164 and 165.) The position of the

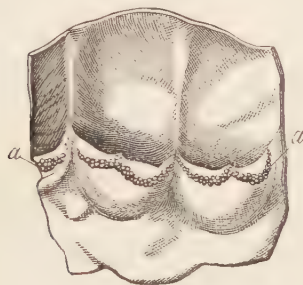


FIG. 164.

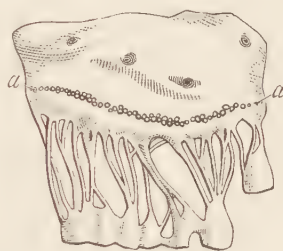


FIG. 165.

FIG. 164.—*Early endocarditis of the aortic*; and FIG. 165, *of the mitral valve.*
—(After Green.)

a, a, bead-like fringe of vegetations.

vegetations on the valves is probably also to some extent determined by the distribution of the blood-vessels. When the endocardium, covering the chordæ tendineæ or walls of the heart, is implicated, vegetation may of course also be seen in these situations.

At first the vegetations are of small size, and give the affected portion of the membrane a beaded or roughened appearance,—as the case progresses they may become larger, and sometimes attain to considerable dimensions, forming fungating projections which may be sufficiently large to produce considerable obstruction of the valvular orifice.² (See fig. 166.)

¹ That mechanical causes exert a very decided influence in determining the maximum position of the inflammation, vegetations, etc., is very clearly seen in some cases of fungating and ulcerative endocarditis,—as Wilks and Moxon, and many other observers have insisted upon. (See figure 168.)

² Fungating vegetations of this description are more common in the ulcerative than in the simple form of the disease.

When the chordæ tendineæ are swollen and covered with vegetations, they very much resemble in shape ears of corn. (See fig. 166.)

Portions of the vegetations are not unfrequently detached and carried by the blood current to distant organs, producing characteristic embolic symptoms.

The subsequent changes vary considerably in different cases. In the majority, there is more or less absorption of the inflammatory products, and cicatricial contraction and hardening of the affected tissues occur. This process of healing is however, very apt to be attended with disastrous results, for in consequence of the cicatricial contraction the normal relationship, of the valve segments to the valvular orifices, is more or less interfered with, and valvular incompetence or valvular obstruction, or both conditions, very frequently remain. Acute endocarditis, in short, is very often the immediate and direct cause of chronic valvular lesions. In some cases the inflamed segments become adherent to one another or (very exceptionally) to the walls of the heart; and this is another way in which valvular stenosis or valvular incompetence may be established. Sometimes the inflamed and softened parts yield to the blood pressure. Valvular aneurisms, valvular ruptures, or ruptures of the chordæ tendineæ may follow. (See figs. 169, 170, and 171.)

The microscopical appearances of endocarditis.—A section through the normal endocardium is composed of the following parts:—

1. A layer of flat endothelium cells. (See fig. 167.)
2. A layer of branched connective tissue cells, and a dense net-work of elastic fibres.
3. A layer of fibrous and elastic connective tissue arranged in the form of trabeculæ. The thickness of this layer varies considerably in different parts of the heart. It is much thicker in the auricles (more especially the left auricle) than in the ventricles, and is doubtless intended to strengthen the comparatively weak muscular wall of the auricles, and to resist the blood pressure. The outer fibrous layer is directly continuous with the sub-endocardial connective tissue, which

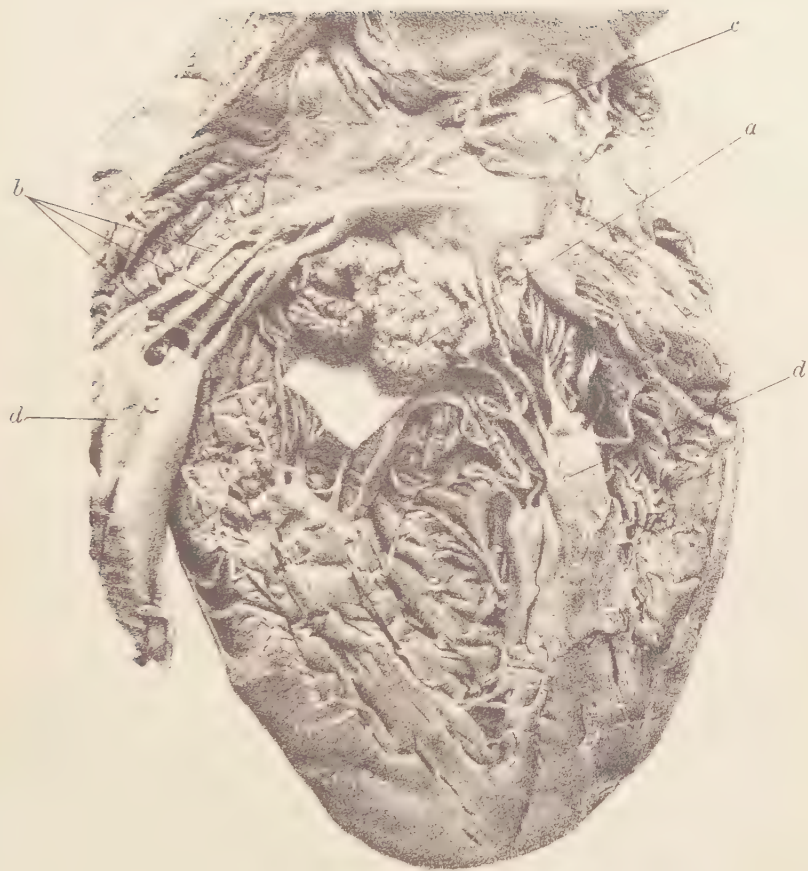


FIG. 166. *Vegetations on the Mitral Valve. (Natural size.)*

A large mass of firm vegetations (*a*) is attached to the free margin of the anterior segment of the mitral valve; many of the chordae tendineae (*b*) are thickened and roughened by deposits of fibrine. A piece of white paper has been placed in the mitral orifice, *i.e.* behind the anterior cusp. The letter *c* points to the base of the aorta and aortic valve cusps; *d, d*, papillary muscles.

in its turn is continuous with the connective tissue of the myocardium.

In the sub-endocardial connective tissue blood-vessels are situated. Fat cells, and unstriped muscular fibres are also seen beneath the endocardium in certain parts of the heart.

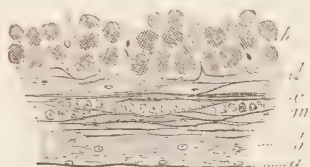


FIG. 167.—*Section through the normal endocardium.*—(After Quain.)

a, Lining epithelium; *b*, connective tissue with fine elastic fibres; *c*, layer with coarser elastic fibres; *d*, subendo-cardial connective tissue continuous with the intermuscular tissue of the myocardium; *h*, muscular fibres of the myocardium; *m*, plain muscular tissue in the endocardium.

The valves are simply folds of the endocardium separated by more or less connective tissue. (See figs. 181, 182, and 183.) The chordæ tendineæ of the mitral valve are attached to the fibrous layer on the ventricular surface of the valve.

Now in acute endocarditis the following changes are observed :—

1. Proliferation of the endothelial layer. This change is seldom seen, but in favourable sections, made in the earliest stages of the affection, it could probably be demonstrated.

2. Proliferation of the branched cells of the second layer. This is one of the most striking features of acute endocarditis, and it is to the proliferation of the cells of this layer that the swollen appearance of the valves is in great part due.

3. Infiltration of the whole thickness of the valves with leucocytes, and proliferation of the connective tissue corpuscles.

4. A deposit of fibrine, in the meshes of which leucocytes become entangled, on the surface of the inflamed part.

The vegetations, which are the characteristic naked eye features of acute endocarditis, are therefore seen to be composed of an organised base formed by the cellular proliferation

of the second layer, on the top of which a deposit of fibrine has taken place.

During the process of absorption and cicatrisation many of the cellular elements disappear; others become elongated and spindle-shaped, and are ultimately converted into fibrous tissue; the whole tissue becomes dense and firm, and calcareous particles are often deposited at the seat of the lesion.

In a considerable number of cases, endocarditis is combined with pericarditis; the naked eye and microscopical appearances, characteristic of inflammation of the pericardium are then of course seen.

Inflammation of the endocardium does not seem to have the same tendency to produce serious myocarditis as inflammation of the pericardium has; nevertheless the two conditions (endocarditis and myocarditis) are not unfrequently associated; and in severe cases of endocarditis the layer of muscular fibres next the endocardium is generally involved. In almost all cases there is some inflammatory infiltration of the fibrous septa and lymphatic spaces between the muscular fibres adjacent to the inflamed membrane. When the chordæ tendineæ are affected, the papillary muscles may become seriously implicated.

The Clinical History of Acute Simple Endocarditis.

Symptoms.—In a large proportion of cases, acute simple endocarditis (when uncomplicated with pericarditis or myocarditis) is entirely unattended by cardiac symptoms,¹ the

¹ The statement in the text which represents my own experience, accords with that of most authorities. It is important, however, to mention that Sibson differs from the generally received opinion. He states, 'in nearly every case of endocarditis the patient presents great or considerable general illness. Thus in sixty-two of the seventy-one cases of mitral endocarditis the illness was great or considerable, in two it was definite, and in five it was slight; while in two there is no description of the general state of the patient.' . . . 'Those cases in which there was no endocarditis, present a very different aspect, since in scarcely one-third of them was there considerable general illness.' . . . 'The illness in cases of endocarditis is peculiar. It differs from and is superadded to that due to simple rheumatic inflammation of the joints, and is such as to call the attention of the physician to the state of the heart. The face may be flushed all over, the forehead, nose, lips and chin being of as high a colour as the cheeks, a state that is usually associated

clinical picture being made up of the symptoms due to the primary disease. In the course of acute rheumatism, for example, acute endocarditis may become developed, and may run its course without any præcordial pain, palpitation, or distress, without, in short, any subjective cardiac sensations being complained of, and without any evident mechanical derangement of the circulation. The pulse and temperature, which are of course always more or less affected by the rheumatic attack, may present no characteristic modifications. The patient may recover from the rheumatic fever, totally unconscious that his heart has been seriously involved; and it is perhaps only when the symptoms of a valvular lesion are subsequently, and perhaps a long time afterwards, developed, that the occurrence of rheumatic endocarditis is suspected. These are the cases of so called *latent* endocarditis.

In a *second* group of cases, there is more or less præcordial uneasiness and palpitation; with the onset of the endocardial inflammation, the pulse becomes quicker and more irritable, and the pyrexia already present, as the result of the rheumatic condition, may become distinctly increased. There are in short some subjective cardiac sensations, but no symptoms due to mechanical derangement of the circulation. A large proportion of cases comes under this head.

with profuse perspiration, drops of sweat standing in beads on the surface—a condition, however, that may be present in cases with severe affection of the joints without endocarditis. Thus when endocarditis exists, the face loses the brightness, glow, and smoothness, and the variety of hue and tone of health, and becomes clouded, being dusky, dull, or ashy in hue, or glazed, or unduly white, or even of a bluish tint. The countenance, no longer expressive of interest in things and persons around, or even of pain in the limbs, is marked by internal trouble. The aspect of the patient is altered, often profoundly so, being anxious, depressed, or indifferent. The eye loses its lustre and expression, and becomes heavy and dull. The breathing is usually affected, being more or less quickened. Pain in the region of the heart, sometimes severe and lasting, sometimes slight or transient, amounting perhaps only to uneasiness, was present in about one-fourth of the cases of tricuspid and mitral murmur belonging to the earlier series, and in one-half of the later series treated by rest.’—*Russell Reynolds*, vol. iv. p. 471.

The reader is referred to the original for a full description of the symptoms, which Sibson thinks are characteristic, space only allows me to give the more important, and those which were most frequently present.

In a *third* group, the endocarditis may be latent, or the symptoms characteristic of the second group of cases may be present, when symptoms indicative of embolic plugging of some distant vessels (such, for example, as right hemiplegia with aphasia) suddenly occur, and at once direct attention to the heart.

In a *fourth* group of cases cardiac symptoms (both subjective cardiac sensations and symptoms due to mechanical derangement of the cardiac pump) are prominent. I must repeat, however, that the cases included in this group constitute a minority, probably a small minority of the whole.¹ In this group are included those cases in which:—

(a) The valvular lesion resulting from the endocarditis is so severe as to seriously interfere with the normal course of the circulation during the acute stage of the attack; as, for instance, where ulceration or rupture of a valve segment occurs, or where the vegetations are produced in such exuberance as to seriously obstruct the valvular orifice;² or where thrombi are formed in the cardiac cavities.

(b) Acute endocarditis attacks a valve which is already in a state of disease, upsetting the balance of compensation, and producing serious symptoms.

In treating of the general pathology of cardiac affections, I laid particular stress upon the fact, that so long as the valvular lesion is compensated by secondary hypertrophy, cardiac symptoms are usually slight or wanting. It must, however, be remembered, that in cases of this description the heart is working up to its full power; it has, as it were, little or no reserve force with which to meet sudden emergencies; under such circumstances an endocarditis, which in a healthy person (*i.e.* a person whose heart was previously healthy) would be unattended by cardiac symptoms, may be sufficient to upset

¹ I am of course speaking of acute simple endocarditis, not of the ulcerative variety of the disease.

² All of these conditions are rare in simple endocarditis. Ulceration and rupture of the valves are much more common in the infective than in the simple form of endocarditis. Obstruction of the valvular orifices by vegetations is of theoretical rather than practical importance, and is more likely to occur in the ulcerative form.

the balance, and to produce serious indications of cardiac failure (palpitation, shortness of breath, congestion of the lungs, dropsy, etc.)

(c) The endocarditis is complicated with pericarditis (endo-pericarditis), or with myocarditis (endo-myocarditis). In cases of this description, in which cardiac symptoms are often very prominent, the clinical picture is of course a complicated one. A severe attack of endo-pericarditis is attended with symptoms similar to those previously described (see page 311); while in endo-myocarditis, palpitation, irregular and tumultuous action of the heart, a quick, weak, irregular pulse, dyspnœa, dropsy, and other symptoms of cardiac failure and of mechanical interference with the course of the circulation, are apt to arise.

Physical signs.—The physical signs of acute simple endocarditis are somewhat uncertain; and it is in consequence of this fact that opinions differ so widely as to the frequency with which the condition occurs in acute rheumatism.

Inspection, palpation, and percussion fail, as a rule, to give any definite information, and it is only when the inflammatory changes are sufficiently severe to produce valvular incompetence,—when, in short, the normal heart sounds are replaced by murmurs,—that the condition can, with any approach to certainty, be recognised.

We have seen that the mitral valve is more frequently and more severely attacked than the aortic; we further know that incompetence is much more readily produced at the mitral than at the aortic orifice; hence it will be easily understood that a systolic murmur at the apex, which indicates mitral regurgitation, is the physical sign which is most frequently met with in these cases. It must, however, be confessed that the exact value of this physical sign (*i.e.* of a systolic apex murmur) as an indication of acute endocarditis is by no means settled; and as the question is one of great practical importance, I must consider it in some detail. I shall limit my remarks to rheumatic endocarditis, which includes the great majority of cases.

Most observers will, I suppose, agree, that in a large proportion of cases of acute rheumatism in which the heart was previously healthy, a systolic apex murmur, in other words, mitral regurgitation, is developed; and the great practical question which we have to consider is, what is the value of that murmur as an indication of acute endocarditis?

Now mitral regurgitation arising under such circumstances may theoretically be due to:—

1. Organic changes in the valve flaps, the result of endocarditis.

2. Relative or muscular incompetence, the result of rheumatic myocarditis.

3. Relative or muscular incompetence, the result of muscular debility, induced by the febrile process, independently of rheumatic myocarditis.

4. Relative or muscular incompetence, the result of anæmia, which is always to a greater or less extent developed in the course of a case of acute rheumatism,—and which may of course be present before the attack commenced.¹

Now in trying to determine which of these is the true cause of the condition, the argument, which has been so ably advanced by Dr Sansom, as to the period of the attack at which the murmur is first observed is, I think, of the greatest importance.² He has directed attention to the fact, that in rheumatic fever the systolic apex murmur is generally developed early in the attack, whereas in other febrile diseases, such as typhoid, typhus, etc. (in which mitral regurgitation, due to febrile changes, so to speak, in the cardiac muscle, is common, and in which endocarditis is rare), the systolic apex murmur is developed late in the attack. He, therefore, concludes, and I think with great justice, that

¹ I am speaking of the ordinary rheumatic fever of adults. In children, and sometimes also in adults, acute rheumatism is so mild as not to be attended by anæmia. Nevertheless in these cases, more especially in children, there is a strong tendency to endocarditis. This argument may be added to those which will afterwards be advanced against the anæmic origin of the systolic apex murmur which so frequently appears in the course of rheumatic fever.

² *Lettsomian Lectures*, p. 18.

the cause of the mitral regurgitation, which appears early in acute rheumatism, is not identical with the cause of the mitral regurgitation which develops late in typhoid and typhus,—that it cannot in short be due to simple febrile changes¹ in the cardiac muscle.

For the same reason it cannot be anæmic. The murmur is in fact developed before the anæmia. I am speaking of the general run of cases. An attack of acute rheumatism may of course occur in a person who is anæmic (in fact it would appear that anæmia is a predisposing cause of acute rheumatism), and in such a case, the mitral murmur might be anæmic, and not endocarditic. But an apex systolic murmur due to anæmia would in all probability be accompanied by a well-marked basic (pulmonary) systolic murmur. As a matter of fact, however, the mitral systolic murmur which occurs in the early stages of acute rheumatism, is not, as a rule, preceded or accompanied by a pulmonary systolic murmur. Further, we know that in the later stages of acute rheumatism, *i.e.* at the period of the attack when the anæmia does become developed, pulmonary systolic murmurs are frequently observed.²

We are obliged, therefore, to conclude that the mitral regurgitation, which accompanies the earlier stages of acute rheumatism, must be due to some condition or conditions peculiar to the rheumatic attack. We also know that endocarditis and myocarditis do frequently occur in acute rheumatism, that in fact murmurs of the kind we are at present considering often become permanent, and are found after death to be associated with organic mitral disease.

It is reasonable to conclude, therefore, that the regurgitation is due to one or other or both of these conditions (endocarditis or myocarditis).

It is much more difficult to solve the second part of the problem, and to come to a positive conclusion as to whether

¹ I am of course aware that M. Hayem and others consider the changes in the cardiac muscle, which occur in typhoid and typhus, inflammatory in nature. What I mean to emphasise is, that they are not of the same character as the rheumatic changes.

² *Sibson, Russell Reynolds' System of Medicine*, vol. iv. p. 489.

the regurgitation is due to organic changes in the valve flaps, the result of endocarditis, or to 'relative' or 'muscular' incompetence, the result of myocarditis.¹

It is, I think, quite open to doubt whether the slight organic changes, which we see in the mitral valve after death in some cases of acute rheumatism in which a systolic apex murmur was present during life, could have been the cause (*i.e.* the mechanical cause) of the regurgitation. Inflammation of the valve cannot of course produce regurgitation except by producing softening or yielding of the flaps, or by leading to the formation of vegetations sufficiently extensive to prevent the perfect adaptation of its edges.²

Further, recent observations go to show, that in these cases the inflammatory changes are not limited to the parts of the valves which appear diseased to the naked eye; but that a corpuscular infiltration extends all through the endocardium and even between the muscular fibres of the heart.

Again, we know that vegetations may be present on the mitral valve flaps, and yet there may have been no regurgitation, as evidenced by a systolic apex murmur during life.

For all these reasons it is difficult to exclude the influence of the myocardium. We must, I think, allow that the regurgitation is not in all cases due to mechanical changes produced in the valve flaps by inflammation of the endocardium, but that in some cases, if not in all, the inflammatory changes in the myocardium (more especially in the mitral sphincter and the papillary muscles) play an important part in its production.

But this admission does not, in my opinion, detract from

¹ It is probably impossible to give a positive opinion on the point. What we want is a series of *post-mortem* examinations, with careful microscopical observation of the condition of the valves and muscle in the earlier stages of acute rheumatism. It is only by this means that we can hope to determine :—

(1) Whether in all cases in which endocarditis is present, there are also changes in the myocardium.

(2) Whether endocardial changes are present or not in all cases in which a mitral murmur is observed in the early stages of acute rheumatism.

² *Sibson, Russell Reynolds' System of Medicine*, vol. iv. p. 475.

the importance of the murmur as a sign of endocarditis. All the evidence, which we at present possess, goes, I think, to show that endocarditis very generally accompanies, if it does not cause,¹ the inflammatory changes in the myocardium to which I have just alluded.

It has also been argued that since in many of these cases the murmur disappears, endocarditis could not have been present; for, it is said, when a valve is once attacked with endocarditis and rendered incompetent, the valvular changes never disappear.

Such a conclusion is, I conceive, untenable.

In the *first* place, it is impossible in most cases to determine by any clinical test with which we are acquainted, whether the regurgitation will disappear or whether it will remain; in other words, whether the derangement of the heart will or will not terminate in organic disease. To decide the question, as it usually is decided, by the result, and to say that in those cases in which a valvular lesion remains there was endocarditis, and in those cases in which no valvular lesion remains there was no endocarditis, is by no means satisfactory. It would, in my opinion, be much more logical to conclude that the same morbid process was present in both cases; that in some cases the inflammatory changes were slight, and were (so far as clinical examination can detect) completely recovered from; while in other cases they were more severe, and were followed by structural changes, and the usual physical signs of valvular disease.

In the *second* place, I believe, in opposition to the generally received opinion, that inflammatory changes affecting the mitral valve often do result in complete recovery so far as the functions of the valve are concerned; in other words, that in many cases of acute rheumatism in which an apex systolic murmur was present during the attack, and subsequently disappeared leaving the heart, so far as clinical observations could detect, perfectly healthy, the mitral valve

¹ The inflammatory process, in some cases at all events, seems to spread from the endocardium to the adjacent fibrous tissue between the muscular fibres.

was actually affected by endocarditis during the rheumatic attack.¹

I see no reason why valvular changes sufficient to produce slight incompetence (such, for example, as corpuscular infiltration of the valve segments and adjacent parts of the myocardium, and small vegetations) should not be absorbed, so as to allow the valve to become competent again. Indeed both clinical and pathological experience is, I think, in favour of such a view. It must be remembered that all degrees of endocarditis occur. After death slight thickenings of the mitral valve are often found in the bodies of persons who have previously suffered from acute rheumatism, but who did not present any mitral murmur previous to death; in such cases, endocarditis was, I suppose, present during the rheumatic attack, but the valve had afterwards become completely competent. Again, persons who have recovered from acute rheumatism with, it is said, healthy hearts (*i.e.* in whom a mitral murmur, if it did exist during the earlier stages of the attack disappeared during convalescence) are more liable to be affected with mitral valve disease in after life than other members of the community; and one cause of their greater liability to subsequent disease is probably to be explained by supposing that slight valvular changes (insufficient to produce regurgitation, and therefore unattended by a murmur) remained as the result of rheumatic endocarditis, which in after years formed the starting point, as it were, for serious organic disease.

The evidence which is afforded by the examination of cases of chorea, and which I have stated below in the form of propositions, very strongly corroborates the view advanced above. It is as follows:—

¹ I do not say that the mitral regurgitation directly resulted from these valvular changes; it may have been due to an associated affection of the 'mitral muscle.' This question has already been discussed. I do however say, that whatever the cause of the regurgitation (whether mechanical changes in the valve segments the result of endocarditis, or dynamic changes in the mitral sphincter the result of myocarditis) the strong probability is, that the mitral valve was affected with inflammation, and that the inflammatory changes did not progress and lead to a permanent valvular lesion, but that the recovery was complete, so far as the functions of the valve were concerned.

1. In a large number of cases of chorea a systolic mitral murmur is present.

2. In the majority of these cases the murmur completely disappears, leaving the heart perfectly healthy so far as can be ascertained by clinical tests.

3. In the vast majority of cases which happen to prove fatal, either during or shortly after an attack of chorea, a fringe of bead-like vegetations is found on the mitral valve. This change is found not only in those cases which die from the chorea, and in which there were cardiac symptoms, but in cases which die from accidental or other complications, and in which there were no cardiac symptoms; in cases, in short, which, so far as the heart symptoms and physical signs are concerned, differ in no respect from those which recover without any cardiac valvular disease remaining.

4. In those cases in which the patient recovers from chorea and dies *after an interval*, the valve is generally found to be perfectly normal. 'It is only,' says Dr Sturges, 'where death supervenes in the course of chorea or very shortly afterwards, that this appearance is met with, and in those cases not always.'¹

From this evidence we are, I think, warranted in concluding, that in cases of chorea which present the physical signs of mitral regurgitation, a bead-like fringe of vegetations is generally present on the mitral valve during the stage of chorea, and that it subsequently disappears (*i.e.* is absorbed).

Further, we may, I think, conclude that the vegetations in chorea are indicative of endocarditis, and that they are strictly analogous to the vegetations which appear on the valves in rheumatic endocarditis; in fact, in many cases the choreic endocarditis is undoubtedly rheumatic.

If then choreic endocarditis with vegetations can be completely recovered from, why cannot rheumatic endocarditis with vegetations be recovered from?

The disappearance therefore of the mitral murmur does not, in my opinion, prove that endocarditis was not present. Further, if we grant, as is probably the case, that the mitral

regurgitation which is present in so many cases of acute rheumatism, is in part at least due to the condition of the cardiac muscle (the corpuscular infiltration of the myocardium) we can still more easily understand how on the subsidence of the inflammation, and the absorption of the inflammatory products, the muscular closure of the valve may again be perfectly effected, and the murmur may disappear.¹

The conclusions therefore to which I would come, with regard to the value of a systolic mitral murmur in acute rheumatism are as follows:—

1. That a systolic mitral murmur occurring in the early stages of a first attack of acute rheumatism, in a person free from previous valvular disease and not previously anæmic, shows that there is a rheumatic affection of the heart.

2. That the mitral regurgitation in such cases may be due either to mechanical alterations in the valve segments, resulting from endocarditis, or to muscular incompetence, the result of a rheumatic affection of the muscle of the heart, or to a combination of the two conditions.

3. That although it is impossible in many cases, to determine which of these conditions (valvulitis or 'muscular' incompetence) is the exact cause of the regurgitation; the strong probability is, that the endocardium is affected.

I would therefore regard a murmur of this description as very strong evidence of acute endocarditis.

4. That in exceptional cases the murmur (in the early stages) is due to anæmia. In these cases there would probably be a history of well marked anæmia; a basic systolic (pulmonary) murmur would also be present.

5. That a mitral systolic murmur originating in the later stages of acute rheumatism may be due to a rheumatic inflammation of the heart (endocarditis or myocarditis);

¹ Dr Sansom directs attention to the fact, that in these cases the subsidence of the murmur must not be taken as proof of the cure of the endocarditis; for in some of them, at all events, the process of absorption and cicatrisation continues to progress, the valvular orifice becomes deformed, and the murmur indicative of established organic disease appears.

anæmia; or simple muscular relaxation such as is met with in all fevers.

But, further, it has been shown, more particularly by the late Dr Sibson, and by Sir Wm. Gull and Dr Sutton, that the mitral systolic murmur is in many cases preceded by a prolonged first sound; that the mitral regurgitation is in fact, as we would naturally expect, not abruptly but gradually established; and that for some time, it may be for some days, before a murmur can be distinctly heard, the first sound at the apex is prolonged, impure or murmurish. These writers are, therefore, of opinion, and with their opinion I entirely agree, that a prolonged or impure first sound in the early stages of acute rheumatism is highly suggestive of endocarditis. 'Prolongation of the first sound,' says Sibson, 'is the first whisper of an approaching murmur, the last of a departing one. . . . We must look then upon prolongation of the first sound as a sign of actual or probable or threatened inflammation of the heart.'¹

In addition to these physical signs, others are sometimes present, which, more particularly in severe cases, make the diagnosis still more certain.

Endocarditis affecting the aortic valve may produce aortic regurgitation. In such cases the valve flaps are generally covered with extensive vegetations, and are not unfrequently ulcerated. A double aortic murmur is then present, and is proof positive of serious organic disease.²

Accentuation of the pulmonary second sound, the evidence of secondary changes in the right heart, such as increased dulness on percussion, due to rapid dilatation; signs of pulmonary congestion or mechanical derangement of the systemic venous circulation—when developed in the course of acute rheumatism, and independently of any complication in the lungs capable of seriously obstructing the pulmonary circulation—are of course the strongest possible evidence

¹ *Russell Reynolds' System of Medicine*, vol. iv. p. 493.

² A systolic aortic murmur may be due to anæmia, but a diastolic murmur is always organic.

of extensive mitral regurgitation ; and it would be quite safe to predict in any case in which extensive mitral regurgitation was suddenly developed in the course of rheumatic fever, that endocarditis with or without myocarditis was present.¹

In addition, Dr Sansom states that in some cases he has observed reduplication of one or other of the heart sounds as an early sign of endocarditis, and so far as his experience enables him to judge, the endocarditis in cases of this description has been followed by stenosis rather than by regurgitation.²

Complications.—In a large proportion of cases of acute endocarditis there is probably some inflammatory infiltration of the myocardium, more especially of the fibrous septa between the muscular fibres immediately adjacent to the endocardium. It is only, however, in a small number that the muscular substance of the heart is permanently affected.

Pericarditis is a more common complication than severe myocarditis—for, according to Sibson's statistics, every third case of rheumatic endocarditis is complicated with pericarditis.³

Accidental complications due to emboli, such as hemiplegia, enlargement of the spleen, etc., are not uncommon.

It would be out of place to describe here the numerous diseased conditions which may be associated with endocarditis, and which depend upon the nature of the primary condition, in the course of which the endocarditis occurs, rather than upon the endocarditis itself. The albuminuria and dropsy, which so frequently occur in association with scarlatinal endocarditis, may be instanced as an example.

¹ In other words, acute myocarditis rarely if ever produces *per se* extensive mitral regurgitation in a case of acute rheumatism. Under such circumstances endocarditis is, I believe, almost invariably present.

² *Lettsomian Lectures*, p. 18.

³ In a total number of 326 cases of acute rheumatism observed by Sibson, endocarditis was present without pericarditis in 108 cases, and with pericarditis in 54.—*Russell Reynolds*, vol. iv. p. 187.

Onset, Duration, and Terminations.

Onset.—The onset of uncomplicated endocarditis is, as a rule, insidious; it is only in a small proportion of cases that there are any subjective sensations which direct attention to the heart.

Duration.—It is difficult to give any definite opinion as to the duration. The physical signs are uncertain, and, fortunately, our opportunities of determining the point by *post-mortem* investigation are comparatively rare. The duration of the apex systolic murmur, in those cases in which there is good reason to believe that the mitral regurgitation is associated with endocarditis, is probably our best guide, though a very imperfect one; for, on the one hand, the inflammatory process must have made considerable headway before the valve becomes incompetent, and on the other, the valve may probably regain its competence some time before the acute changes have subsided.

Terminations.—It is probable, I think, that in a considerable number of cases in which the inflammatory changes are slight, the products of inflammation are for the most part, if not entirely, absorbed, the valve regains its competence, and a practical cure is effected.

In many cases, if not in all, some thickening of the valve segments remains, and in some cases these thickenings become, in after life, the starting points of chronic valvular lesions.

It must, however, be admitted that in the majority of well-marked cases the termination is more unfavourable. The process of absorption and cicatricial contraction in a considerable proportion of cases produces such structural alterations, that stenosis or incompetence of the valvular orifice, or a combination of the two conditions, results. In many cases, in short, a chronic valvular lesion remains. These valvular lesions are, as we shall afterwards¹ see, of all degrees of severity, but even in the mildest cases (*i.e.* in

¹ The future progress of these cases will be considered under the head of chronic valvular lesions.

those cases in which there are for a time, it may be for years, no symptoms of cardiac derangement), the ultimate termination is very generally unfavourable.

In a minority of cases the condition terminates fatally during the acute or subacute stages of the disease, with all the clinical symptoms and signs of an advanced valvular lesion (see 440).

Diagnosis.—When a murmur is heard over the præcordial region in a case of acute rheumatism or other affection, in the course of which acute endocarditis is apt to arise, we must of course endeavour to determine whether the murmur is actually indicative of inflammation of the endocardium or not.

The steps in the diagnosis are as follows;—

1. Is the murmur endocardial or exocardial?
2. If endocardial, is it due to endocarditis?
3. If inflammation of the endocardium is present, is that inflammation of the simple or ulcerative form?

Step No. 1.—*Is the murmur endocardial or exocardial?*

There is very seldom any difficulty in deciding whether the murmur is produced within or without the heart. In those cases in which the difficulty does arise,—and, according to Dr Sansom it is chiefly in children that this is likely to occur,—the question can usually be determined by reference to the points of distinction which have already been detailed. (See table III. p. 330.)

Step. No. 2.—*If the murmur is endocardial, is it due to inflammation of the endocardium?*

In attempting to answer this question, we have in the first place to determine whether the murmur is an old one or a recent one. In those cases in which the condition of the heart, prior to the attack for which the patient comes under observation, is unknown, it is sometimes difficult or impossible to determine this point. We have to rely chiefly upon the physical examination of the heart, aided to some extent by

the previous history of the case. There is little difficulty in those cases in which well-marked secondary changes in the heart, such for instance as hypertrophy of the left ventricle, or hypertrophy and dilatation of the right ventricle, are present. Accentuation of the pulmonary second sound, with a minor degree of dilatation of the right heart, is also suggestive of an old valvular lesion; but the evidence is then by no means so conclusive, for acute dilatation of the heart is sometimes observed in endocarditis (rarely however in the early stages of the condition); and mitral regurgitation, sufficiently free to be attended with considerable increase of the blood-pressure within the pulmonary artery, and, therefore, with well-marked accentuation of the pulmonary second sound, is of frequent occurrence during the acute stage of endocardial inflammation.

A history of shortness of breath on exertion, of swelling of the feet, or of other well-marked cardiac symptoms, is very strong evidence of old disease.

The occurrence of a previous attack or attacks of acute rheumatism is also suggestive of old organic disease.

It must, however, be remembered that old valvular disease does not exclude recent endocarditis, on the contrary, it rather favours its development. In all cases of acute rheumatism, in which we are satisfied of the existence of an old valvular lesion, we should strongly suspect the presence of recent inflammation; and in cases of this description, in which the cardiac symptoms undergo notable and sudden aggravation during the rheumatic attack, the presence of acute endocarditis is almost certainly indicated. In those cases in which the murmur is a recent one (*i.e.* has developed during the present attack), and in which the symptoms and signs of valvular lesion are rapidly developed, there is of course no difficulty. The occurrence of a presystolic mitral or tricuspid, or of a diastolic aortic or pulmonary murmur, under such circumstances, is quite conclusive, whether the murmur is attended with cardiac symptoms or not, for these murmurs always depend upon mechanical changes in the valve flaps or valvular orifices.

There is much more difficulty when, as is usually the case, the murmur is systolic. I have already detailed in full the reasons which lead me to regard a mitral systolic murmur or impure first sound, *occurring in the early stages of an attack of acute rheumatism*, and in a person previously free from cardiac disease or marked anæmia, as highly suggestive of acute endocarditis.

The same murmur (a mitral systolic murmur) when it occurs in the *later* stages of the attack, is possibly anæmic in character, more especially if the patient is markedly anæmic, and if the development of the mitral murmur is preceded by a pulmonary systolic murmur; but it is impossible to give a positive opinion on the point. The murmur may be organic; and the wise physician, while hoping for the best (*i.e.* that it is anæmic), will take every precaution, and treat the patient as if the murmur depended upon organic disease.

A basic systolic murmur, aortic or pulmonary (but especially the latter), which develops late in the attack, and when the patient is anæmic, is very generally functional.

Murmurs which are loud and well propagated are generally organic, musical murmurs are always so.

Step No. 3.—If inflammation of the endocardium is present, is that inflammation of the simple or ulcerative form?

The consideration of this question must be deferred until the ulcerative form of endocarditis has been described. (See page 411.)

Prognosis.—In forming the prognosis the following points must be taken into account:—

(1) The nature and severity of the primary affection with which the endocardial inflammation is associated.

(2) The state of the heart itself.

(3) The condition of the other tissues and organs.

In rheumatic endocarditis, the immediate result is usually favourable, but some cases die, as we have seen, during the acute or subacute stages of the disease.

Symptoms indicative of grave mechanical derangement of

the circulation, and of failure of the heart, such as dyspnœa, dropsy, cyanosis, a quick, weak, and irregular pulse, are very serious indications. Cases in which aortic regurgitation is acutely established are, as a rule, more serious than those in which the mitral valve is affected.

Pericarditis, and more particularly myocarditis, add to the gravity of the attack. The existence of previous valvular disease, other things being equal, materially increases the danger.

The ultimate prognosis is in a large proportion of cases unfavourable, for many patients, who recover from the acute affection, ultimately suffer and die from chronic valvular disease. The prognosis should always, therefore—even in the mildest cases—be guarded, for so long as the slightest indications of endocarditis are present, it is impossible to be certain that serious valvular lesions may not remain. The disappearance of the systolic apex murmur, which is the most common physical sign of endocarditis, is, of course, the most favourable indication. It is not, however, proof positive of the cure of the endocarditis, for, as Dr Sansom has shown, the regurgitation may have been due to an affection of the myocardium rather than of the endocardium; the corpuscular infiltration of the myocardium may be absorbed; the valve may become competent; but the endocardial cicatrix, so to speak, may continue to contract, and a murmur indicative of organic valve disease may in a short time be established. In all cases, therefore, the heart should be examined from time to time during the period of convalescence. The character of the murmur and the condition of the heart must, of course, be taken into consideration. A soft valvular murmur, which is not well propagated, and which is not attended by any marked accentuation of the pulmonary second sound or by secondary changes in the right heart, very often disappears without leaving any organic change behind. *Vice versâ* the lesion is a serious one in those cases in which the murmur is heard below the angle of the left scapula, in which the second sound is accentuated, and more especially in which there are secondary changes in the right heart (increased dulness resulting from dilatation, a systolic tricuspid murmur, etc.).

A murmur, which continues after convalescence is fully established, may, in exceptional cases, disappear at the end of some months. As a rule, however, such a murmur, and the organic changes on which it depends, are permanent. The extent and severity of the lesion which remains, vary greatly in different cases. Each case must of course be judged on its own merits in accordance with the principles which will be described when I come to treat of chronic valvular lesions.

Treatment.—As in the case of pericarditis, it is necessary to consider both the prophylactic and curative treatment of the affection.

Prophylactic treatment.—Since acute simple endocarditis is seldom primary, but usually occurs in the course of some other disease, our prophylactic measures must be directed :—

(1) *To warding off or preventing the primary affection with which it is apt to be associated.*

Space does not allow me to enter into details as to the manner in which acute rheumatism, chorea, scarlet fever, measles, diphtheria, pyæmia, puerperal fever, and the other affections with which endocarditis is apt to be associated, are to be prevented. The prophylactic of these affections consists, of course, in avoiding the causes—both predisposing and exciting—which produce them. The reader who wishes further information on this subject must consult some of the standard works in which the ætiology of these affections is fully treated of.

(2) *To cure the primary affection, when once it is established, as speedily as possible, and in particular to adopt such a plan of treatment as is most likely to prevent inflammation of the endocardium.*

Applying this indication to the treatment of acute rheumatism, which is, as we have repeatedly seen, by far the most common cause of acute endocarditis, we must endeavour to cut short the rheumatic attack, to allay the pain and fever as speedily as possible, and to enforce a rigid system of rest, for, as Sibson has shown, the relief of pain and suffering, together

with absolute rest, exerts a most beneficial influence in preventing cardiac complications.

Sibson's observations on this point are, I think, of extreme importance, and have a very direct bearing upon the treatment of acute rheumatism by salicin and its compounds. I make no apology, therefore, for quoting what he says in full. He states :—'We here find that in the series of cases of acute rheumatism that were treated by a system of absolute rest, the proportion of those that were attacked with endocarditis was slightly less than that of those that were not so treated. Thus far the comparison is but slightly in favour of the treatment of acute rheumatism by a rigid system of rest ; and this would seem to suggest that a certain and a very large proportion of cases of acute rheumatism are habitually and intrinsically attacked by endocarditis. When, however, we extend the comparison, and ascertain the proportion in which those cases of endocarditis, not previously so affected, acquired permanent valvular disease, so as to injure health during the remainder of life, and to shorten life itself, we discover that the series of cases not treated by a system of absolute rest were thus permanently injured in a far larger proportion of cases, amounting to more than twice as many, or in the ratio of eight to three, than in those that were treated by rest.

'If we pursue the inquiry further, so as to discover the relative extent to which the interior of the heart was inflamed in the two series of cases, we discover that there was but one instance, or 1 in 24, of those with endocarditis and without previous valvular disease, of the series treated by a rigid system of rest, that gave definite evidence of inflammation of both the aortic and mitral valves, while in 19 instances in 127, or 1 in 6·7 of the same kind of cases that were not treated by a rigid system of rest, there was direct evidence of aortic regurgitation. In nine, or rather ten, of those cases that were not treated by rest, there was a mitral murmur, and therefore direct evidence of inflammation of the mitral valve ; but in the remaining nine cases there was also evidence of mitral endocarditis in the shape of a tricuspid murmur, or prolongation of the first sound, with intensification of the pulmonic second sound, and obstacles to the flow of blood through the lungs.¹ The whole chain of evidence points

¹ There will probably be considerable difference of opinion as to the exact value which these physical signs possess as evidence of acute endocarditis. In the absence of any obstruction in the lungs, they are indicative of some embarrassment in the left heart. Now, considering the frequency of acute endocarditis in acute rheumatism, and the frequency with which a systolic mitral murmur and permanent evidence of valve disease spring, as it were, directly out of these physical signs, I am personally disposed to think that when they are met with in the early stage of the attack, and are unassociated with anæmia and basic cardiac murmurs, that they are suggestive of endocarditis, but certainly not a distinct proof of its presence.

then, I think, irresistibly to the conclusion that the extent, severity, and permanent ill effects of the endocarditis were much greater in the series of cases that were not rigidly treated by rest than in the series that were so treated.¹

Now for the relief of the joint affection and the reduction of the temperature, there is, I suppose, a consensus of opinion, that the salicylic treatment is better than any other. Twenty to thirty grains of salicin or salicylate of soda given every two hours, succeed, in a large proportion of cases, in completely removing the joint affection, and in reducing the temperature within forty-eight hours. If then Sibson's observations and deductions are correct, this drug ought to exert a very beneficial influence in preventing cardiac complications. If it does not exert such a beneficial influence, we must, I think, conclude that, while relieving the joint affection and fever, it actually exerts a prejudicial effect on the heart, rendering the endocardium more liable to be affected, and neutralising the protective influence which the relief of the joint affection and fever ought, according to Sibson's observations, to produce.

The most extensive observations which have as yet been published on this point, are those which were brought forward at the great debate before the Medical Society of London in the year 1881; and it seems to have been pretty generally concluded, that the result of the statistics then brought forward was to show that salicin and its compounds do not exert the protective influence in warding off cardiac complications which we would *a priori* have expected.

Personally I have considerable hesitation in accepting that conclusion as correct, more particularly as some of the leading speakers in that debate (Drs Hilton Fagge, Broadbent, Douglas Powell, Havilland Hill, and Herman), seemed, on the whole, to think that the influence of the salicylic compounds in preventing endocarditis was favourable, and also for the following reasons:—²

¹ *Russell Reynolds' System of Medicine*, vol. iv. p. 527.

² I state these reasons with great diffidence, for I am fully aware that it is often extremely difficult to form correct conclusions upon figures and facts with which one is not personally familiar. Those who were present at the debate, and more particularly the speakers who were familiar with *all* the facts (for in analysing

In the *first* place, the result of that debate was almost entirely based upon hospital cases; and, as Dr Douglas Powell very justly, I think, observed, and as Dr Maclagan¹ has pointed out, it is almost impossible to determine this question by the results of hospital practice. In a large proportion of hospital patients, endocarditis—as evidenced by a systolic apex murmur—is already developed on the patient's admission. These cases must, of course, be excluded in any inquiry as to the influence which salicin has in preventing endocarditis. Again, there is good reason for supposing, as I have previously pointed out in detail (see p. 379), that endocarditis is present at the time of the patient's admission, in many cases in which there is no murmur, but in which a bruit subsequently becomes audible. These cases must also be excluded, for, as Dr Maclagan puts it, 'this saving action (of the salicyl compounds) cannot be got unless they are given in adequate quantity *before the poison has begun to act on the heart*.'² It may, of course, be said, and the argument doubtless has some force, that the salicyl compounds should not only prevent, but that they should also allay the endocardial inflammation even after it is developed. There are not the same fallacies in deciding this point, and there seems to be a very general opinion that these drugs have no influence in this direction. Dr Fagge 'fully admits that salicylic acid seems to have no power of controlling or arresting the cardiac complications of acute rheumatism when once they have developed themselves.'³

Dr Maclagan believes that when endocardial complications occur, 'the treatment by salicylate of soda occasionally increases the patient's danger. I refer especially,' he says, 'to those cases more numerous than is usually supposed, in which the muscular substance of the heart is the seat of inflammation. Myocarditis has, for its pathological condition,

such a large number of cases as were analysed by some of the speakers in this debate, it is of course impossible to go into every detail), were, of course, much more likely to come to a more correct conclusion than I can possibly do.

¹ *Rheumatism*, p. 271.

² *Locus cit.* p. 269.

³ *Lancet*, Dec. 17, 1881, p. 1033.

thickening, softening and enfeeblement of the muscular walls, chiefly those of the left ventricle. If to this enfeeblement is added that which sometimes follows the administration of salicylate of soda, the patient's condition is thereby rendered more serious, and the continuous administration of the salicylate might turn the scale against him.¹ It must be particularly noted that Dr MacLagan does not believe that salicin has this depressing influence on the heart. *Dr Broadbent* is so strongly of opinion that salicyl and its compounds are useless in the cure of endocarditis, that the moment he recognises any cardiac inflammation he discontinues their administration.²

In the *second* place, all endocardial murmurs are not indicative of endocarditis, in the later stages of acute rheumatism more especially, when anæmic murmurs are most apt to arise, there may be great difficulty in deciding whether endocarditis is present or not.

In the *third* place, observers are by no means agreed as to the value which is to be attached to different endocardial murmurs as signs of endocarditis. It is difficult, therefore, to compare the results of different physicians who may attach very different values to the same facts. It is still more difficult to compare the observations made to-day with the observations made twenty or thirty years ago.

Although personally I am inclined to attach very great importance to apex systolic murmurs, arising in the *early* stages of acute rheumatism and *without* basic systolic (pulmonary) murmurs, as indicative of acute endocarditis, I think it would be wise to base our inquiry upon the number of cases in which permanent valvular lesions—as to the symptoms and signs of which there is very general agreement—remain, rather than upon the frequency with which endocarditis occurs during the attack. The nature of the valve lesion which remains is also a point of some importance, for, as Sibson has shown, aortic regurgitation requires for its production (as a general rule) a greater degree of endocardial inflammation than mitral lesions.

¹ *Lancet*, Jan. 14, 1882, p. 59.

² *Lancet*, Jan. 28, 1882, p. 138.

There is another reason why endocarditis—as evidenced by an apex systolic murmur—should not be taken as the standard in estimating the value of the salicyl compounds in preventing rheumatic endocarditis; for if, as most observers admit, these drugs (salicin according to Dr Maclagan excepted) exert a special depressing influence upon the heart, their use is very likely to be attended with such relaxation of the cardiac muscle as will produce mitral regurgitation (from muscular or relative incompetence) independently of endocarditis.

For these reasons it is, I think, difficult to decide the question by means of hospital statistics; and more especially to estimate the results by the frequency with which endocarditis is supposed to occur during the attack of rheumatic fever. The inquiry should, I think, be based upon the number of cases in which permanent valvular lesions remain. Cases in which cardiac lesions were known or suspected to have existed previously, or cases in which the patient had previously suffered from rheumatic fever, should be rigidly excluded. The exact nature of the resulting valvular lesion should be stated. Instead of comparing hospital cases in which, as we have seen, endocarditis is so often established before the patient comes under observation, the inquiry should be based on the results of private practice, those cases only being selected in which the patient comes under observation at the very commencement, say within the first twenty-four hours of the attack.¹

To be absolutely accurate, the result would have to be based upon a very large number of cases, all observed by one thoroughly competent, reliable, and unprejudiced observer. But since it is impossible for any single observer to meet with

¹ Cases which come under the care of the practitioner within twenty-four hours of the development of the attack would, as Dr Hilton Fagge pointed out in introducing the discussion on the salicylates before the Medical Society of London, be 'of far more than the average severity, since persons affected with the milder forms of the disease would often wait for a time before seeking medical advice.' That no doubt in some respects is an objection; but the advantages of such a method of inquiry are, I think, so far as our present purpose is concerned, so much greater than those which any other method is likely to afford, that I do not hesitate to recommend it strongly to the profession.

anything like a sufficient number of cases to serve as an adequate basis of results, the inquiry would of course have to be entrusted to a number of observers of known accuracy, and it would be of the utmost importance that they should all make their observations on exactly the same plan.

In the present position of therapeutics we would hardly, I think, be warranted in making a series of investigations with the view of testing the efficiency of different drugs, for I thoroughly agree with Dr Hilton Fagge in thinking, that we would not be justified in withholding salicin and its compounds from our patients at the commencement of rheumatic fever unless there were some special contra-indication to the use of the drug. But we might, I think, determine much more satisfactorily by this means than by any other, the proportion of cases in which permanent valvular lesions follow the salicylic plan of treatment; and this, I believe, is the most important point which we wish to decide with regard to the treatment of acute rheumatism. Having determined the point in the case of the salicylates, we would have a fixed standard with which to compare any other method of treatment which may in the future lay claim to be superior to that which at present we believe to be the best.

The method of case-taking recommended by the Collective Committee of the British Medical Association, should be used, with the following slight additions necessary for the special purpose in hand:—

Method of case-taking in acute rheumatism, with the object of determining the frequency with which permanent cardiac valvular lesions follow the salicylic plan of treatment.

Note A. Only those cases of acute articular rheumatism are to be recorded in which:

- (1) The patient was previously free from cardiac valvular disease.
- (2) The attack of rheumatic fever is a first attack.
- (3) The treatment is put into force within twenty-four hours after the first manifestation of symptoms, *i.e.* of joint pain.

B. In all cases salicin to be used and not salicylate of soda; and the drug to be given after the manner laid down by Dr Maclagan, viz.: 'at least thirty grains every hour till there is decided evidence of action, and then the dose should be diminished slowly.'¹

¹ *Rheumatism*, p. 272.

C. Whenever any of these directions are departed from, the exact nature of the modification introduced must be specified on the card.

D. In addition to the points specified by the Collective Investigation Committee of the British Medical Association, the following particulars must be added :—

Condition of the heart (1) at the end of convalescence ; and (2) six months subsequently, as regards :—

(a) Position of apex beat.

(b) Force of impulse.

(c) Murmurs (their rhythm, points of differential maximum intensity, direction of propagation, and sound characters).

(d) Condition of pulmonary second sound—normal—increased.

(e) Subjective cardiac sensations.

(f) Signs of mechanical derangement of the circulation.

In the treatment then of acute rheumatic endocarditis, I would strongly advise the administration of full doses of pure salicin after the manner recommended by Dr Maclagan, and I cannot help thinking that if this treatment were vigorously carried out in the earlier stages of the attack, the frequency of endocarditis and other cardiac complications would be materially diminished.

Some authorities recommend that full doses of alkali (thirty grain doses of bicarbonate of potash every two or three hours until the urine is alkaline) should be combined with the salicin, and they adduce in support of this plan of treatment the highly favourable results, as regards the cardiac affection, obtained by Drs Fuller, Dickenson and others. Some again, in addition to the salicin, advise the local application of blisters to the joints, as recommended by Dr Davies. Personally I am in the habit of wrapping the affected joints in cotton wool, or cotton wool sprinkled with belladonna and chloroform liniment, as recommended by Sibson. When the pain is very severe a hypodermic injection of morphia should be administered, and repeated, if required, for some hours must necessarily elapse before the beneficial effects of the salicin become apparent. A rigid system of rest must at the same time be strictly enforced, the greatest care being taken to avoid anything, such as mental anxiety, worry, or agitation, which is likely to excite the action of the heart. The patient must of course be

placed in blankets. During the acute stage, the diet must be entirely liquid, and should consist chiefly of milk. The condition of the bowels must also be attended to. I do not, as a rule, prescribe a purge quite at the commencement of the attack; it is better, I think, to wait a day or two until the acuteness of the joint affection has subsided. The patient should not be allowed to leave his room for the purpose of evacuating the bowels, it being of the greatest importance to avoid exposure to cold.

If the salicin fails after a fair trial (four or five days), I would advise the administration of quinine in combination with alkalis. However mild the attack, or however rapid the return to health, the patient should be kept in bed for at least a week after the joint affection and the pyrexia have completely subsided.

During convalescence all sources of cardiac excitement, both bodily and mental, must be carefully avoided. Tonics, more particularly quinine, iron and arsenic, should be prescribed. The greatest care must be taken to avoid exposure, or anything which is likely to bring on a relapse.

These are the chief measures which I would recommend in a case of acute rheumatism uncomplicated by the symptoms or signs of acute endocarditis. But before leaving the prophylactic treatment of rheumatic endocarditis, I must again direct attention to the fact which Sansom, Barlow, and others have so justly emphasised, that endocarditis not unfrequently arises in the course of mild rheumatic attacks, and that in children more especially, in whom the tendency to inflammation of the endocardium is so much stronger than it is in adults, the most trivial rheumatic manifestations should always be carefully attended to. I cannot too strongly endorse Dr Sansom's teaching on this most important point.

The prophylactic treatment of choreic endocarditis.—There are, as I have previously pointed out, ample grounds for believing that endocarditis is of frequent occurrence in chorea. Now, in dealing with cases of chorea, it is of the greatest

importance to keep this fact in view ; for although, as a rule, the endocardial inflammation completely subsides, leaving the heart free from organic change (so far as our means of clinical investigation can detect, and as the future progress of the case amply proves), in some cases valvular lesions are (either immediately or in future life) established.¹

The same general rules as to avoiding all cardiac strain and excitement, exposure to cold, and other conditions likely to act as exciting causes of endocarditis, and which have been already mentioned in speaking of the prophylaxis of rheumatic endocarditis, are equally applicable here ; and it is the more necessary to insist upon this point, since chorea is too generally regarded as a trivial affection. The patient should be kept in bed during the attack. Arsenic, chloral, and bromide of potassium, are the remedies which I have found most useful. The bowels must be carefully attended to ; but I must refer the reader for details of the treatment of chorea to works on general medicine.

The prophylaxis of endocarditis in scarlet fever, measles, diphtheria, puerperal fever, pyæmia, etc.—We know of no special means by which the occurrence of endocarditis can be prevented during the acute stages of these affections, other than the ordinary means of treatment which it is advisable to adopt for the treatment of the primary disease.

It is especially important, as Dr Sansom has pointed out, to direct attention to mild cases of scarlet fever, measles, and other febrile diseases in children, and to protect the patient, more especially during convalescence, from exposure to cold, and the other exciting causes of endocarditis.²

The treatment of the endocardial inflammation when it has

¹ Dr Barlow has suggested that the greater frequency of mitral stenosis in young women than in young men may be due to the fact that girls suffer much more frequently from chorea than boys. Be that as it may, it is, I think probable, that in some cases, at all events, the endocardial changes established during an attack of chorea, and apparently completely recovered from, may afterwards be the starting point of serious valvular disease.

² *Lettsonian Lectures*, p. 33.

arisen.—Theoretically our first indication is to cut short and allay the inflammatory process. As a matter of practice this indication cannot be satisfactorily carried out, for, in the first place, the inflammation has already made considerable progress before it can, with any approach to certainty, be recognised; and in the second place, we cannot bring the depletory and other local measures which are useful in the early stages of most inflammations, to bear with any certainty upon the interior of the heart.

General venesection, the internal administration of tartar emetic, and other remedies of a similar description, which used to be so lavishly employed in the early stages of internal inflammations, are especially out of place here, for one of the most important indications which we have to carry out is to avoid anything which will seriously depress the action of the heart.

As soon as rheumatic endocarditis is distinctly recognised, it is advisable, as Dr Broadbent has pointed out, to discontinue the administration of salicylate of soda. This remedy does not appear to possess any curative action so far as the endocardial inflammation is concerned, and it does seem in many cases to produce cardiac depression. There is not, according to Dr MacLagan, the same (positive) objection to salicin, which may still be continued after the endocardial inflammation is recognised, provided that the joint affection necessitates its use.

It is of the utmost importance to keep the heart, so far as it can be kept, at rest. With this end all bodily movement¹ and mental excitement must be avoided; excessive action or irritability of the heart must be moderated, and for this purpose belladonna and digitalis are the most useful drugs. Unless the joint affection continues, the pyrexia is seldom sufficiently great to require any special means of treatment. For the reduction of temperature salicin should first be tried, and if that drug fails, as it not unfrequently does in the presence of a cardiac complication, recourse must be had to quinine.

In the majority of cases this treatment is all that is

¹ In those cases in which the joint affection still continues, the patient instinctively avoids all bodily movement.

required in addition to the dietetic and other measures suitable to treatment of the primary affection.

In severe cases, when the valvular apparatus is seriously damaged, or when the inflammation of the endocardium is complicated with pericarditis or with myocarditis, other measures may be called for. One of the most important indications in bad—and in fact in all—cases, is to look out for symptoms of cardiac failure, and, when necessary, to strengthen and stimulate the action of the heart. Digitalis, brandy, ammonia, and ether, are the remedies which are most useful for this purpose.

When the right heart becomes seriously embarrassed, local depletion by means of leeches is often most useful. But these measures will be more particularly detailed when I come to treat of valvular lesions. (See p. 474.)

Should thrombi form in the cardiac cavities, the internal administration of ammonia may be tried, as first recommended by Dr B. W. Richardson. Ten to fifteen drops of the liquid ammoniæ may be given internally every two or three hours; or ten grains of the carbonate of ammonia may be administered in a table-spoonful of the solution of the acetate three or four times daily, as recommended by Bartholow.

Embolic and other complications must of course be met by appropriate remedies as they arise. Space does not allow me to go into details here.

ULCERATIVE ENDOCARDITIS.¹

Synonyms.—Septic Endocarditis, Infectious Endocarditis, Diphtheritic Endocarditis, Endocarditis Maligna (Virchow), Arterial Pyæmia (Wilks).

¹ Professor Osler objects to the term 'ulcerative,' because, on the one hand, there may be no actual ulceration of the valves in cases which present the characteristic features of the disease during life, and on the other, endocardial losses of substance may occur without these symptoms. I have retained the term, *firstly*, because it is the title most generally used in this country; *secondly*, because ulceration, though not invariably present, is in most cases a characteristic feature, and represents, so to speak, the highest degree of development of the local lesion; and *thirdly*, because the term represents a mere pathological fact, and does not suggest any theory as to the causation of the disease, as the terms septic, infectious, and diphtheritic do—an important point when we consider the difference of opinion which exists as to the ætiology and pathology of the affection.

Definition.—An acute inflammation of the endocardium, which is characterised, *pathologically*—by the formation of fungoid vegetations and (usually) ulcerations of the endocardium ; *clinically*—by great prostration, frequency of pulse, fever of an irregular or suppurative type, symptoms of a typhoid or pyæmic character, together with enlargement of the spleen, and the local manifestations of embolic infarctions of various organs. The invariable termination, so far as at present known, is in death. Micrococci frequently abound in the cardiac vegetations.

Ætiology.—There is considerable difference of opinion as to the nature and cause of ulcerative endocarditis.

Some observers regard the micrococci, which are met with in the cardiac vegetations, and in many cases also in the embolic infarctions of distant organs, as the cause of the disease, and think that they (the micrococci) gain entrance into the blood from an external wound or an abrasion, or through the gastro-intestinal tract or air passages. There seems to be little doubt that in some cases the cardiac lesion is simply one manifestation of a general pyæmic or diphtheritic condition. In some puerperal cases, for instance, as Rosenstein points out, ‘undoubted diphtheritic affections have been seen in the mucous membrane of the uterus and vagina. And in these very cases the likeness between the diphtheritic matter found on the genitalia and that which covers the endocardial abscess is so strong, and parasitic organisms have been detected with such certainty in both cases, that nothing but the most stubborn incredulity could deny a connection brought about by the blood, between the affection of the genitalia and the endocardial centre.’¹

Others, while admitting that micrococci are very generally, if not invariably, present in the cardiac lesion, think that they are rather the consequence than the cause of the affection, or at all events that their presence is accidental ; in other words, they believe that the cardiac vegetations form a suitable nidus in which the micrococci can develop. In support of this view

¹ *Ziemssen's Cyclopædia*, vol. vi. p. 70.

they state that micrococci are frequently found in the vegetations of rheumatic endocarditis. Osler,¹ for example, who is one of the most powerful advocates of this view, states:— ‘Micrococci are not peculiar to the vegetations of the ulcerative form of endocarditis, but exist in the small beadlike outgrowths of the rheumatic and other varieties of the disease, as Klebs was the first to point out. My experience tallies with his; in seven specimens of verucose or plastic vegetations which I have examined all contained micrococci. . . . So far as my observation goes, the micrococci do not exist in the blood during the course of the malady. Nor are they constantly found in the infarcts. The occurrence of micrococci in the warty vegetations of rheumatic endocarditis, and in the extensive ulcerative outgrowths so frequently met with in old sclerotic valves, are facts strongly opposed to the view of their specific poisonous nature. The micrococci appear to be identical in these cases, though Klebs states that those of rheumatic endocarditis are larger and have a brownish tint. I cannot say that these differences have been constant in the specimens which I have examined. It seems a pertinent question to ask, if in the malignant form of endocarditis, the micrococci are so potent, why in other cases in which they are equally prevalent, should they be inert? Of course it may be urged that the micrococci may be of different kinds or possess diverse qualities, or that the resistance offered by the tissues to their penetration varies in different cases, or that it is only in weakened and debilitated states that those little bodies thrive. There is, I think, something worthy of attention in this latter view. If we study the conditions under which endocarditis develops, we find almost invariably that the patients are the subject of some other constitutional affection, which, as we say, predisposes to it. What determines the precise form of the endocarditis, we do not know, but the soft endocardial vegetations form a suitable nidus for the development of micrococci. They appear in fact to be just as much normal components of endocardial outgrowths as the fibrin

¹ *Transactions of the International Medical Congress of London, 1881*, p. 345.

fibrils, which are usually deposited and among which the micrococci abound. It is evident that these structures are common elements in a series of endocardial processes which display totally different symptoms and arise under different conditions. How far they are responsible either for the development of the endocarditis or for the subsequent characters which, in the grave form it assumes, the evidence does not, I think, warrant as yet a very positive opinion.¹

To sum up, ulcerative endocarditis does not appear to be a specific infectious disease in the same sense that typhoid, scarlet fever and smallpox are, for, so far as I am aware, inoculation experiments have failed to reproduce the disease. In some cases the endocardial lesion appears to be a manifestation of a general pyæmic or diphtheritic condition. In these cases, an unhealthy wound, diphtheritic ulcer, or diseased patch of mucous membrane, is the original source of infection, to which the cardiac lesion is secondary, though the infective emboli, which become detached from the cardiac vegetations and ulcerations, will of course aggravate the pyæmic condition.

In other cases the cardiac lesion is the primary local source through which the system becomes impregnated. This group includes cases originating in the course of acute rheumatism and old standing valvular disease, and in it we may provisionally include those cases in which the origin is obscure, and in which the primary cause of the endocarditis is unknown. To these cases the term 'arterial pyæmia,' proposed by Dr Wilks, seems very appropriate. In them, owing to a depraved condition of the vital forces, and of the heart in particular, the resisting power of the tissues is diminished. On the occurrence of endocarditis (as the result of rheumatic or other causes) micrococci, which we must suppose are always circulating in the blood, but which in a state of health are unable to multiply and establish themselves in the tissues, find a suitable nidus, and develop first in the cardiac vegetations and subsequently in distant organs and parts. In these cases,

¹ *Transactions of the International Medical Congress of London, 1881*, p. 346.

the cardiac lesion is the local source of infection through which the blood becomes poisoned and the pyæmic condition established.

It is probable, as Dr Murchison has suggested, that, in some instances in which the symptoms of ulcerative endocarditis are observed, the local lesion is not cardiac, but is situated in some part of the arterial system.

The following are the chief conditions with which ulcerative endocarditis appears to be associated :—

1. *Acute or subacute rheumatism.*—The exact frequency with which this association occurs has yet to be determined. Rosenstein's view that the majority of cases of ulcerative endocarditis occur in the course of acute rheumatism does not appear to be correct, for Osler found, on analysing the reports of fifty-seven cases, that in fifteen only, or 26.3 per cent. was any mention made either of acute rheumatism or of previous rheumatic attacks.

In the rheumatic cases there is almost invariably, in addition, a previous history of ill health or depressed condition of the system such as follows want, exposure, the abuse of alcoholics, etc.

2. *Old standing cardiac disease* (valvular lesions, fibroid thickening of the cardiac walls).—It is probable that in many of these cases, the endocarditis is also rheumatic. A depressed condition of the system seems in these cases also to be an important factor in determining the form which the endocardial inflammation ultimately assumes. The low organisation and defective vascularity of chronically diseased valves probably also favour the production of ulceration, as Virchow was the first to point out.

3. *Pyæmia, puerperal fever, diphtheria.*—There seems to be no doubt, as I have already mentioned, that in some cases the endocardial ulceration is only one manifestation of a pyæmic condition, which has its original source of infection at the periphery in the shape of an unhealthy wound, ulcerated or diphtheritic mucous membrane, etc. In the majority of cases, however, in which pyæmic symptoms are associated with endocarditis, the sequence of events is probably different, the

cardiac lesion being the primary event to which the infection of the whole system is secondary.

4. *Injuries unattended with local suppuration.*—Osler has described a case in which ulcerative endocarditis followed a fractured leg (simple fracture unattended with local suppuration), and in the cases which he analysed he found a considerable number in which there was a history of some injury or wound. In cases of this description, in which it is clearly proved that the local wound or injury did not act as a local source of pyæmic infection, we can only suppose that the injury had a depressing influence on the system which favoured the production of the ulcerative rather than the simple form of endocarditis, the endocardial inflammation being probably due to some other condition or conditions.

5. *Syphilis.*—In some of the recorded cases the patient suffered from syphilis before the onset of the attack. Possibly, therefore, in some of these cases the endocarditis was in the first instance syphilitic, and in consequence of the depraved condition of the system, and low resisting power of the tissues, it assumed the ulcerative form.

In addition, there remain a considerable number of cases in which the cause of the condition is obscure.

Age, sex, and occupation.—Like the simple form of endocarditis, and like acute rheumatism, the affection seems more common in young than in old people. Males, because of their being more exposed to depressing external influences, alcoholic excesses, etc., are probably more liable to be attacked than females, though many writers state that the liability to the affection is equal in the two sexes. Persons whose occupations necessitate exposure to cold, want, and other injurious external influences, probably suffer more frequently than other people, but no particular trade seems to predispose to the disease.

Pathological anatomy.—In the majority of cases the left heart is the chief or only seat of the lesion, but the right heart is much more frequently affected than in simple endocarditis.

As in the simple form of inflammation, the valves are the

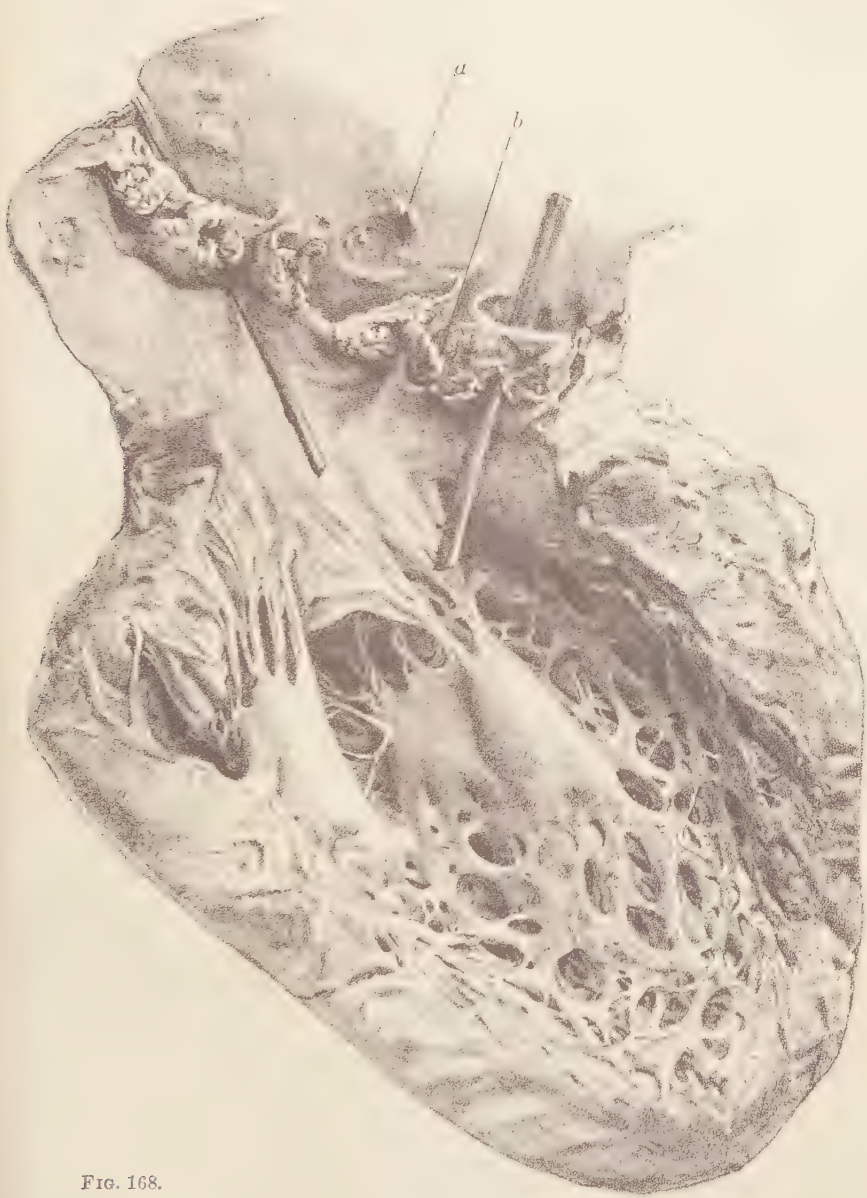


FIG. 168.

Aortic Valve in a case of Ulcerative Endocarditis. (Natural size.)

The Aortic Cusps are thickly coated with vegetations; two of the segments are ulcerated through (pieces of whalebone being placed in the apertures); the letter, a, points to a small depression (commencing aneurism), surrounded by minute vegetations, at the base of the Aorta; it has evidently been produced by the vegetation, b, which at every systole would be forcibly washed against the base of the Aorta at this spot.

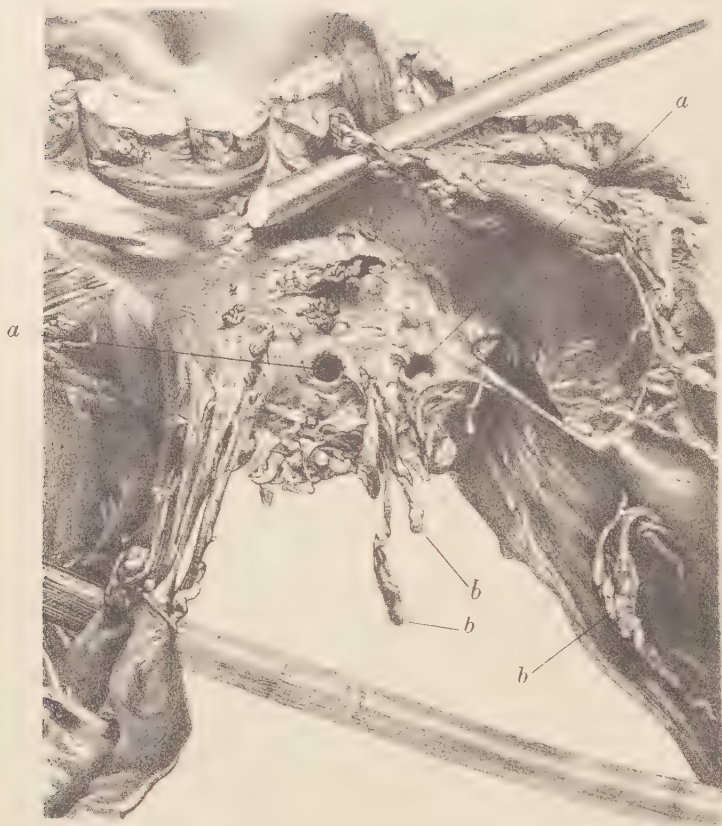


FIG. 169.—*Ulcerative endocarditis; Rupture of the chordæ tendinæ; Aneurisms on the mitral valve. (Natural size.)*

The specimen came under my notice in the *post-mortem* theatre of the Royal Infirmary of Edinburgh, during the winter of 1883-84. The patient was under the care of Professor Greenfield, with whose kind permission the specimen is reproduced here.

A piece of cane has been passed through an ulcer in one of the aortic cusps; *a, a*, point to two ulcers in the anterior flap of the mitral valve, which communicate with the aneurisms seen in fig. 170; *b, b*, ruptured chordæ tendinæ. The mitral and aortic valve segments are covered with numerous vegetations.



FIG. 170.—Aneurisms on the mitral valve, and rupture of the chordæ tendineæ. The same preparation represented in fig. 169. (Natural size, seen from behind.)

a, a, aneurisms; *b, b*, ruptured chordæ tendineæ; *c, c*, vegetations and clots attached to the chordæ and papillary muscles.

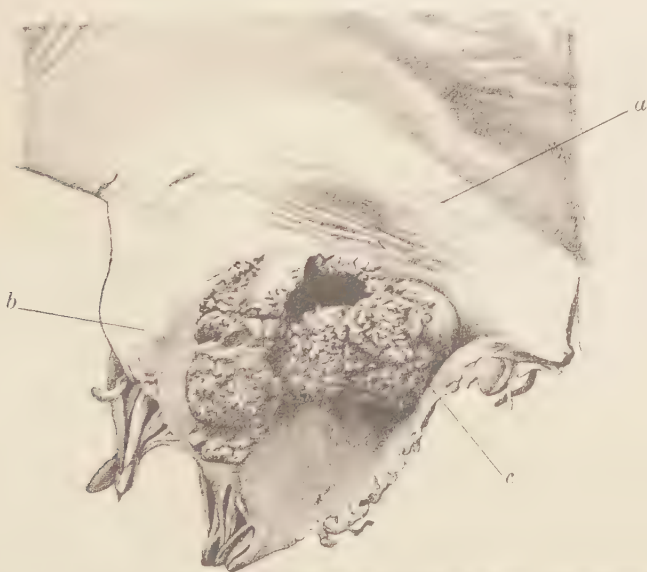


FIG. 170' —Aneurism of the mitral valve, with rupture of the valve cusp. Seen from the auricular surface. (Half as large again as the natural preparation.)

a, wall of left auricle; *b*, anterior segment of the mitral valve, the chordæ tendineæ have been cut short; *c*, aneurism. A large triangular opening is seen in the valve segment.

The ventricular surface of the same preparation is shown in fig. 171.

(Copied by Professor Turner's permission, from a specimen in the Anatomical Museum of the Edinburgh University.)

immense numbers of micrococci, often in the form of ball-like masses (see fig. 173) are embedded.

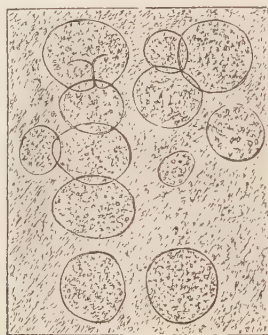


FIG. 173.—Ball-like masses of Micrococci. (*After Osler.*)

The edges of the ulcers or erosions, for in many cases they are superficial, are uneven and granular. The base of the ulcer has usually a dirty greyish-yellow appearance, and to it small blood-clots often adhere. The subjacent tissues are always more or less extensively implicated. The surrounding portions of the endocardium are studded with projecting granulations and vegetations, in many cases, in fact, the ulcers are hidden beneath the fungating growths which surround them.

When an ulcer involves a valve flap, the resisting power of the membrane is of course destroyed, and under the force of the blood-pressure an acute aneurism or bulging of the valve is not unfrequently produced. The valvular aneurisms vary in size, but are seldom larger than a marble. When the anterior segment of the mitral valve is affected, the aneurismal sac bulges into the auricle, the orifice of the sac being situated on the ventricular surface of the valve, *i.e.* the surface on which the blood presses during the contraction of the left ventricle. (See figs. 169, 170, 171, 172.) Aneurisms of the aortic segments bulge into the cavity of the ventricle, the orifice of the sac being situated on the upper or arterial surface of the segment; and here again the direction which the sac takes is determined by the blood pressure. The aneurismal



FIG. 174.

FIG. 174.

FIG. 174. Section through a segment of the aortic valve and adjacent parts of the aorta and heart, showing vegetation on the aortic valve, magnified about 10 diameters. (The aorta has become detached from the auricle.) The recesses: *a*, recess of the aorta; *b*, aortic valve segment; *c*, *c*, vegetation; *d*, wall of ventricle; *e*, wall of auricle; *f*, vegetation on particles in the inner coat of the aorta; and *g*, calcareous particles in the base of the aortic valve.

FIG. 174. — Colonies of micrococci from a case of Ulcerative Endocarditis. (After Osler.)

sac not unfrequently ruptures, and a perforation of the valve segment is produced.

In some cases, more especially in the case of the aortic valve, the free margins of the flaps are more or less completely destroyed by the ulcerative process.

When the ulcer is situated on the surface of the endocardium lining of the cavities of the heart, different results may follow in accordance with its position, extent, and depth. In some cases, an acute aneurism or bulging of the heart wall, or of the septum ventriculorum, occurs. In rare cases the cardiac wall or septum is completely eroded, and a rupture of the heart or a communication between its different cavities (between the two ventricles for instance, when the ulcer is situated on the septum ventriculorum) is established. In other cases again, in which an abscess forms around the base of the ulcer, the inflammatory process may make its way right through the muscular wall of the heart, inflammation of the pericardium being ultimately produced.

An ulcer at the root of the aorta usually results in the production of an acute aneurismal dilatation of the vessel at the weakened spot. An aneurism of this description is generally of small size. It may project into the pericardium or into the pulmonary artery. Occasionally its rupture is the immediate cause of death.

In addition to the cardiac lesions which I have just described, other morbid appearances are almost invariably present. Enlargement of the spleen is always observed ; the organ is in some cases soft and pulpy, similar to the enlarged spleen of fevers ; in others it contains infarctions. The portion of the capsule corresponding to the infarctions is not unfrequently thickened and coated with a layer of recent lymph.

Embolic infarctions and their results (softening, inflammatory deposits, etc.) are almost always found in some of the peripheral parts (*i.e.* the parts peripheral to the heart, such as the spleen, kidney, brain, skin, etc.). In fact, one of the most characteristic features of the disease, both in its pathological and clinical aspects, is the frequent occurrence of embolic infarctions—a tendency which is at once accounted for by the

facts that the vegetations are very friable and easily broken down, and that products of ulceration are washed away by the blood stream as it passes over the floor of the ulcers. The particles which are thus detached are for the most part small, and are therefore carried to the smaller arteries and capillaries, where they stick, and give rise to local inflammations and septic deposits, in which micrococci can often be detected on microscopical examination. In some cases larger portions of the vegetations are detached, and the larger arteries, such, for instance, as the middle cerebral, are obstructed.

Inflammation of the serous membranes, more especially pleurisy and empyema, are not uncommon.

The membranes of the brain are sometimes inflamed; and extensive hæmorrhages have been met with both in the substances of the brain and on its surface. Aneurismal dilatations of the cerebral blood-vessels have also been observed.¹

The mucous membrane of the intestines is sometimes inflamed and ulcerated. Inflammatory deposits in the subcutaneous tissues and purpuric eruptions on the skin, both of which are probably due to embolic plugging, also sometimes occur.

Acute croupous pneumonia is, according to Osler, a frequent complication. The exact relationship of the pneumonia to the endocarditis is not definitely known, but both conditions are in all probability due, as Osler suggests, to one and the same cause. Small abscesses in the heart, probably embolic, are also observed in some cases.

Clinical History.—Different cases of ulcerative endocarditis present great varieties in regard to their individual symptoms and to the rapidity of their course; it is extremely difficult, therefore, to give a general description applicable to all.

The *onset* is, as a rule, sudden. A patient, for example, who is suffering from a mild attack of rheumatic fever, or

¹ All of these lesions—the meningitis, which more frequently involves the hemispheres than the base of the brain, the cerebral hæmorrhages, and the aneurismal dilatations of the cerebral blood-vessels,—are probably the result of emboli.

who is the subject of old cardiac disease, or whose general health has for some time previously been below *par*, without perhaps any definite indications of disease or local lesion, is suddenly seized with a rigor which is quickly followed by fever and grave constitutional symptoms, which I shall presently describe. Occasionally, though very rarely, the onset is gradual. A remarkable case of this description is described by Bristowe,¹ in which for several weeks before the patient's admission to hospital she suffered from what appeared to be intermittent fever.

Symptoms.—Subjective cardiac sensations are, as a rule, slight or altogether absent, though palpitation, præcordial distress, or actual pain in the region of the heart, are occasionally complained of.

In some cases, marked shortness of breath, independently of any obvious pulmonary cause; more or less dropsy; extreme pallor of the countenance; great rapidity of the pulse (*i.e.* symptoms due to deranged action of the cardiac pump), together with characteristic physical signs of cardiac disease (such as—a diastolic aortic murmur—a systolic mitral murmur with accentuated pulmonary second sound and evidence of engorgement of the right heart, etc.), are so prominent that, notwithstanding the presence of fever and other grave constitutional symptoms, which are characteristic of the typhoid or pyæmic types of the affection, the attention of the observer is at once directed to the heart.

In these cases, as indeed in all types of the disease, the spleen is more or less enlarged, and generally tender to the touch; and symptoms due to embolic infarctions in the kidney (such as albuminous or bloody urine and tenderness over the loins), in the brain² (paralysis, delirium, convulsions,

¹ *British Medical Journal*, 1880, vol. i. p. 800.

² The nervous symptoms which are of such frequent occurrence in these cases may be due to a great variety of different lesions, all of which are probably in the first instance due to embolic infarction of the cerebral vessels. Amongst these the following are some of the chief:—Embolic plugging of numerous small vessels; embolic plugging of large vessels, as for instance the left middle cerebral; extravasations of blood into the substance or on the surface of the brain; inflammation of the cerebral membranes.

headache, coma), in the skin and subcutaneous cellular tissue (purpuric symptoms, local swelling and tenderness), or in other parts—are very generally observed and constitute extremely striking and characteristic features of the case. Pneumonia, pleurisy and empyema are common complications. I am in the habit of describing these cases, in which cardiac symptoms and signs are prominent, as the *cardiac type* of the disease.¹

In the majority of cases, the cardiac symptoms and signs are altogether thrown into the back ground by the severity of the general and constitutional symptoms. Fever, usually of an intermittent or markedly remittent type, great prostration and depression, marked increase in the rapidity of the pulse, rigors, profuse perspirations, enlargement and tenderness of the spleen, enlargement of the liver, symptoms due to embolic infarctions and secondary inflammations in the kidney, brain, skin, and serous membranes, and indications of general blood-poisoning, are some of the symptoms which are most frequently observed. Lung complications, such as pneumonia, œdema, etc., often occur. The symptoms sometimes very closely resemble those of typhoid fever, and cases have been repeatedly sent into hospital in which this mistake in diagnosis has occurred.

In these cases, which are described as the *typhoid type* of the disease, the fever is a prominent symptom (ranging from 101° up to 105°, 106°, 107° F., or even higher), and is of a remittent rather than of an intermittent type. Vomiting, diarrhœa, distention of the abdomen, tenderness and gurgling in the right iliac fossa, and eruptions of a roseolar and purpuric character, and in some cases resembling more or less closely the characteristic eruption of typhoid fever, have all been observed. The spleen is found on examination to be enlarged and tender to the touch. The symptoms of cardiac disease are not prominent, and may be altogether absent.

¹ Although it is convenient for descriptive purposes to divide cases of ulcerative endocarditis into certain definite clinical groups, and to describe cases of a *cardiac*, *typhoid*, *pyæmic*, and *agueish type*, the reader must not forget that this division is to a certain extent arbitrary. There is no sharp line of demarcation between these different groups, which run into one another in all directions.

The physical examination of the heart may throw light upon the case, as, for instance, in those cases in which undoubted evidence of organic valvular disease (such, for example, as a diastolic aortic murmur), is detected in a person, whose heart is known to have been previously healthy. But in many cases the information derived from the examination of the heart is much less certain. In some cases in which extensive cardiac lesions have been found after death, no murmur was detected during life. In others, and these perhaps constitute the majority, an apex systolic murmur which, as we have previously seen, may depend upon many different conditions, and which is therefore somewhat uncertain, as a sign of organic valvular disease, is the only physical alteration to be detected. The uncertainty—as to the significance of the murmur—is still further increased if the condition of the heart prior to the present attack of illness is unknown. In cases of this description, the pulse is often very much accelerated, the tongue becomes dry and brown, sordes may cover the lips and teeth, there is often more or less delirium, in many cases the patient, for some time previous to death, lies in an apathetic or semi-conscious condition, the evacuations being passed involuntarily. In short, the general symptoms are those of the typhoid state. In these cases, death is usually preceded by coma.

In other cases of ulcerative endocarditis, the symptoms are identical with, or resemble more or less closely those of pyæmia.¹

In these, which are described as the '*pyæmic type*' of the disease, the fever is intermittent, and presents the irregular ups and downs which are so often met with in connection with internal suppuration, or rather with the absorption into the blood of repeated doses of pus or poisonous materials produced from pus. The temperature may, in brief periods of time, range between 105°, 106°, 107°, 108° F., or even higher on the one hand, and 97°, 96°, 95° F., or lower on the other ;

¹ I have previously pointed out that in some of these cases the cardiac lesion is a secondary manifestation of pyæmia, and that in others the pyæmic condition follows, and depends upon the endocardial inflammation.

and repeated variations of this description may occur in the course of the twenty-four hours. Rigors and profuse perspirations are very prominent in these cases. The complexion is usually pale and sallow, or it may be slightly jaundiced. Cardiac symptoms and signs, and symptoms due to embolic infarctions, similar to those which have been previously described in speaking of the other types of the disease, are also met with.

In exceptional cases the attacks of fever occur at regular intervals, and are characterised by a cold, hot, and sweating stage (the '*ague type*' of the affection). Dr Murchison, for example, relates the case of a patient with disease of the aortic valves, who, for three months before his death, had daily paroxysms of fever, sometimes commencing with a definite rigor, and always ending in copious perspirations, and whose friends were so satisfied that the fever was ague, that nothing would satisfy them until the case had been treated with large doses of quinine.¹

Duration and termination.—The duration of different cases varies considerably. In some, death occurs within a few days from the commencement of the attack; in others, as for example the case mentioned by Murchison, and the one represented in figure 168, the symptoms progress much more slowly, and may continue for one, two, or even three months. The termination, so far as is at present known, is invariably fatal.

Diagnosis.—From the description I have given of the clinical history of the affection, the reader will readily understand that in some cases the diagnosis is attended with considerable difficulty.

Cases of the '*cardiac type*' are, as a rule, readily recognised. The two conditions with which they are most frequently confounded are:—

1. Acute simple endocarditis.

¹ *Lancet*, May 3, 1879, p. 618.

2. Chronic valvular disease, complicated with a specific fever or local inflammation.

The differential diagnosis of acute simple and of ulcerative endocarditis.—The two conditions cannot always be distinguished, for cases are sometimes met with which seem to be connecting links between the two forms. In typical cases there is of course no difficulty.

In ulcerative endocarditis, the rheumatic indications are much less frequently present than in the simple form of the disease; while the general constitutional symptoms are usually much more severe. The physical signs undergo more rapid alterations, and evidences of grave organic disease—such, for example, as a diastolic aortic murmur—are more quickly developed. The fever is, as a rule, higher, and is much more prone to assume the suppurative type. The prostration and rapidity of pulse are much greater. Rigors are common in the ulcerative, but rare in the simple form of endocarditis.

Enlargement and tenderness of the spleen, albuminous or bloody urine, though they may occur in the simple form, are vastly more frequent in the ulcerative variety.

In the simple form, embolic infarctions, are comparatively seldom met with, and usually obstruct large vessels; while in the ulcerative variety showers of emboli are apt to occur, and it is the smaller vessels which are generally plugged.

Typhoid and pyæmic symptoms are common in the ulcerative, but rare in the simple form.

Simple endocarditis, when uncomplicated, is seldom fatal; while ulcerative endocarditis, so far as is at present known, always terminates in death.

The differential diagnosis of ulcerative endocarditis and of chronic valvular disease, complicated with a specific fever or local inflammation.—When a local inflammation or fever is actually present, in addition to the symptoms and signs of cardiac valvular disease, or in a patient who is known to have been previously affected with a chronic valvular lesion, the

diagnosis may be very difficult or impossible. In attempting to decide the point, we must ask ourselves whether a local inflammation and an old cardiac lesion would satisfactorily account for the symptoms, not forgetting that local inflammations, such as pneumonia, for example, are frequently present in ulcerative endocarditis. We have in fact to weigh carefully all the facts of the case, and without committing ourselves dogmatically to either view—for the evidence in cases such as we are at present considering seldom justifies such a course—endeavour to determine to which side the balance of probabilities and weight of evidence incline.

Even after we are satisfied that acute endocarditis is actually present, we have still to determine whether the case is one of ulcerative endocarditis, complicated with a fever or local inflammation, or of simple endocarditis associated with similar conditions. This point we must endeavour to determine in the manner described above.

The differential diagnosis of ulcerative endocarditis and of typhoid fever.—Cases of ulcerative endocarditis are, as I have previously pointed out, often mistaken for typhoid fever. In trying to determine whether a doubtful case is one of the typhoid type of ulcerative endocarditis or of true typhoid fever, the following points must be taken into account:—

1. *The mode of onset and rapidity of course.*—Ulcerative endocarditis, as a rule, begins more abruptly, and progresses more rapidly than true typhoid.

2. *The character of the temperature chart.*—In ulcerative endocarditis, the steady and gradual rise by stages, which is so characteristic of typhoid fever, does not occur. At the height of the disease (ulcerative endocarditis) the remissions are usually much greater than in typhoid, and intermissions, in which the temperature falls to or below the normal, not unfrequently occur.

3. *The temperature pulse ratio.*—In the earlier stages of typhoid the pulse may not be at all increased in frequency, although the temperature is considerably above the normal. In the earlier stages of ulcerative endocarditis there is usually

a marked increase in the frequency of the pulse, quite independently of the amount of the pyrexia.

This point, which is one of considerable diagnostic value in the earlier stages of the disease cannot be relied upon afterwards, since in the later stages of typhoid the pulse frequency becomes greatly increased.

4. *The condition of the heart.*—In the great majority of cases of ulcerative endocarditis there are physical signs of valvular disease even in the earlier stages of the attack. In typhoid fever, mitral regurgitation is frequently met with in the later periods as the result of relative or muscular incompetence, but valvular lesions are rarely present at the commencement.

The exact character of the valvular lesion is also of importance, a mitral systolic murmur developed under observation is much less significant of ulcerative endocarditis than an aortic diastolic murmur developed in a similar manner.

Two sources of error must of course be remembered, viz. :—(1) The cardiac lesion may be an old one complicated with typhoid. (2) Acute endocarditis does sometimes, though very rarely, occur in the course of typhoid. But notwithstanding these sources of error, the presence of a recent valvular lesion is strongly in favour of ulcerative endocarditis.

5. *Diarrhœa, with pea-soup stools, tenderness and gurgling in the right iliac fossa, rose spots on the abdomen,* are on the other hand strongly in favour of typhoid; they do occasionally occur as isolated symptoms in ulcerative endocarditis, but are rarely associated together in the same case.

6. *Symptoms of embolic infarctions* are very strongly in favour of ulcerative endocarditis.

7. *Rigors* are much more common in ulcerative endocarditis than in typhoid; the *prostration* in the earlier stages of ulcerative endocarditis is usually more marked.

The differential diagnosis of ulcerative endocarditis and of pyæmia.—Ulcerative endocarditis may result from pyæmia, and pyæmia may follow ulcerative endocarditis. But since the termination in both of these conditions is, so far as we at

present know, always in death, the point is one of little practical importance, and need not therefore detain us.

It is of great importance, however, to remember that constitutional symptoms identical with the symptoms of pyæmic ulcerative endocarditis are not unfrequently met with in cases of abscess of the liver or other large internal and localised collections of pus.

Cases of this description are, as a rule, distinguished without much difficulty. Attention is to be directed to the condition of the heart on the one hand, and of the liver and other internal viscera, liable to be affected with suppurative inflammation, on the other. The occurrence of embolic symptoms is very strongly in favour of ulcerative endocarditis. It must, however, be remembered that it is not always easy or possible to distinguish embolic infarctions and secondary pyæmic abscesses, which may, of course, be developed in the course of hepatic abscess and other internal suppurations.

The differential diagnosis of ague and of ulcerative endocarditis.—Although cases of ulcerative endocarditis do occasionally occur in which the fever intermits at regular intervals, there is little difficulty in distinguishing them from cases of true ague. The intermissions are seldom so regular; the constitutional symptoms between the attacks of fever are, as a rule, much more considerable, and progressively and quickly increase; quinine is powerless to arrest the paroxysms; cardiac murmurs are almost always present; showers of emboli are apt to occur; and the termination in ulcerative endocarditis is in death.

Prognosis.—The prognosis is quite hopeless; all cases, so far as we at present know, end fatally.

Treatment.—Quinine, in full doses, together with free stimulation, should be tried, and the usual treatment for pyæmia adopted. The treatment recommended for severe cases of simple endocarditis may also be carried out.

CHRONIC ENDOCARDITIS.

Definition.—Chronic inflammation of the endocardium.

Ætiology.—In some cases chronic endocarditis follows acute or subacute endocarditis; in others, the endocardial inflammation is developed very slowly, gradually and insidiously (*i.e.* is chronic from the first), and it is not until grave valvular defects have become established that its presence is suspected or recognised.

In a considerable proportion of cases—more especially when the mitral valve is the seat of the lesion—the endocardial inflammation is of rheumatic origin.

In others, the endocardial changes—which for the sake of convenience we are in the habit of describing as due to chronic inflammation—are closely allied to, or are identical with atheroma. In cases of this description, the aortic valve suffers much more frequently than the mitral.¹

Alcoholic excess, syphilis, and gout, are all predisposing causes; indeed there seems to be little doubt that in some cases the endocardial inflammation is directly due to gout, and some observers think that it is sometimes directly due to syphilis.

But of all the causes of chronic endocarditis, strain is one of the most important—chronic inflammation of the aortic valves is, in some cases, caused by strain;² strain is one of the great causes of atheroma and atheromatous valvular lesions, while the all important influence, which increased cardiac effort, *i.e.* strain, has in perpetuating rheumatic inflammation of the mitral valve has already been insisted upon.

Morbid Anatomy.—The essential feature of chronic endocarditis is the production of a dense tissue, consisting of spindle

¹ These two causes of chronic valve disease are very frequently combined. A valve, for example, which is thickened by the atheromatous process, is often attacked with chronic inflammation.

² *Vide* the aortic regurgitation which is frequently met with in young puddlers, ‘strickers,’ and others who follow laborious occupations, in which sudden, violent muscular efforts are required.

cells and fibres (see figs. 176 and 176'), in the midst of which calcareous particles are frequently deposited. As in the case of acute endocarditis, it is the valvular apparatus of the left heart (segments, annular rings, chordæ, and papillary muscles), which chiefly suffers. In some cases, the valvular orifice is contracted or narrowed by sclerotic changes in the annular ring at the base of the valve. In others, the thickened and rigid segments, which may be fixed together and adherent at their edges, form unyielding projections which narrow the valvular aperture. In almost all cases—whether the orifice is stenosed or not—the loss of elasticity in the valvular apparatus (segments, basal ring, chordæ, or papillary muscles), prevents the perfect closure of the valvular aperture, and incompetence, therefore, results. In short, the practical effect of chronic endocarditis is to produce incompetence, or both stenosis and incompetence (very rarely stenosis without incompetence), of one or other or both of the valvular apertures of the left heart; in other words, to produce a chronic valvular lesion.

But since valvular imperfections, more especially valvular incompetence, frequently result from other causes than chronic endocarditis, it is now desirable to consider chronic valvular lesions as a whole.

CHRONIC VALVULAR LESIONS.

Definition.—Under the head of chronic valvular lesions it is convenient to consider all valvular defects, other than those which have been already described in treating of acute endocarditis. (I shall, of course, describe the chronic valvular lesions which so frequently follow acute and subacute endocarditis, but I shall not repeat what has been said with regard to these lesions during their acute stages.)

Ætiology.—By this definition a great number of different pathological conditions are included under the head of chronic valvular lesions, viz. :—

1. *The chronic valvular lesions due to congenital defects and malformations, and to diseases of intra-uterine life.*—In some of these cases the defect or malformation is so severe as to cause

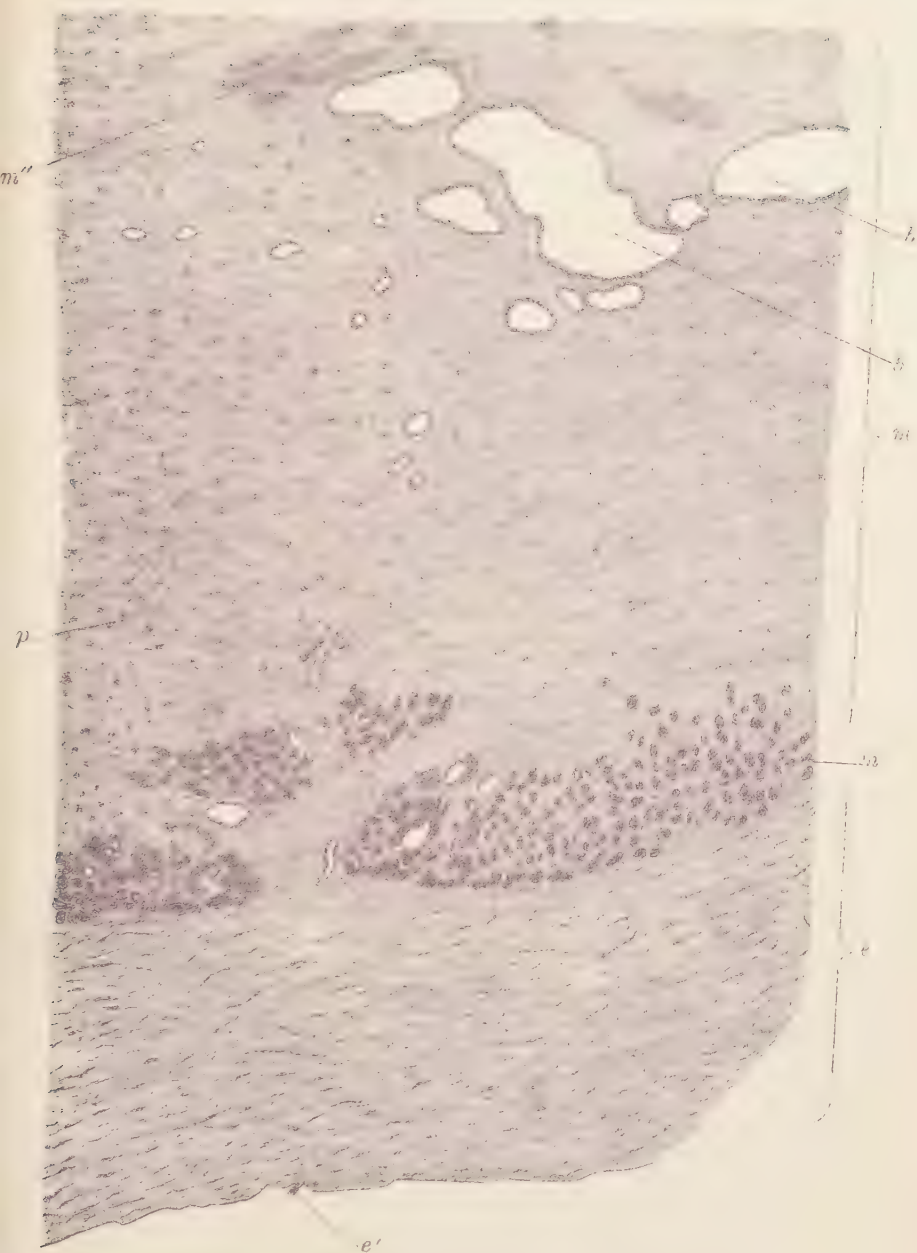


FIG. 176.—Section through the endocardium and adjacent portion of the myocardium in chronic endo-myocarditis. (Magnified about 50 diameters.)

e, the thickened endocardium (the part to which the letter *e'* points is more highly magnified in fig. 176'); *m*, part of the myocardium; *m'*, a band of muscular fibres adjacent to the endocardium which still remains; *m''*, muscular fibres in the centre of the cardiac wall; *p*, pigment granules (the remains of atrophied muscular fibres) lying in the midst of fibrous tissue; *b, b'*, large blood-vessels.



FIG. 176'.—*Chronic Endocarditis.* A part of the preparation represented in fig. 176, more highly magnified ($\times 250$).

a, free surface of endocardium; *b*, *b*, spindle cells; *c*, *c*, corpuscles.

serious symptoms from the date of birth. In others, the condition is so trifling as to be unattended for years, or possibly for the whole period of life, by any injurious effects. It must, however, be remembered, as Dr Peacock has pointed out, that malformed valves are more liable to be attacked by endocarditis than naturally formed ones.

The more severe forms of congenital valvular lesions are very generally right-sided, and are usually accompanied by other abnormalities in the heart, such as a deficiency in the septum ventriculorum, a patent foramen ovale, etc. Stenosis of the pulmonary artery is by far the most common congenital valvular lesion.

2. *The chronic valvular lesions produced by traumatic causes, such as violent effort or external injury.*—In most of these cases, the valve, which gives way, is not absolutely sound at the time of the accident, but is weakened by previous disease (endocarditis, atheroma, etc.). Traumatic ruptures due to strain are in the great majority of instances confined to the aortic segments, and produce incompetence (not stenosis) of that orifice.

3. *The chronic valvular lesions which result from chronic endocarditis (either chronic from the first, or following acute or subacute endocarditis).*—In the majority of cases the lesion is left-sided; and the mitral valve is much more frequently affected than the aortic. A combined condition of stenosis and incompetence usually results. Simple incompetence (*i.e.* incompetence without stenosis) sometimes follows; simple stenosis (*i.e.* stenosis without incompetence) is rare.

4. *The chronic valvular lesions due to atheroma.*—Here again the lesion is almost invariably left-sided, the aortic valve being much more frequently involved than the mitral. A combined condition of stenosis and incompetence usually results. Incompetence of the aortic orifice occasionally occurs without stenosis, but stenosis without some incompetence is very rare.

5. *The valvular imperfections which result from 'relative' and 'muscular' incompetence,* as I am in the habit of terming regurgitation due to simple dilatation or defective muscular

closure of the valvular orifices. Lesions of this description affect the auriculo-ventricular valves (both the mitral and tricuspid) much more frequently than the sigmoid.

Auriculo-ventricular regurgitation may theoretically be either 'relative' or 'muscular,' but as a matter of fact I believe the two conditions are very generally, if not invariably, combined. In cases of this description, unless there is some other valvular lesion present, such for example, as stenosis, the result of chronic endocarditis, the regurgitation is simple, *i.e.* unattended by stenosis. The mitral and tricuspid regurgitation which occurs in chlorosis, progressive pernicious anæmia, exophthalmic goitre, the advanced stages of fevers, etc., are typical examples of this form.

Aortic incompetence is never, of course, 'muscular.' Relative incompetence of the aortic orifice is simple, *i.e.* unaccompanied by stenosis. Increased aortic tension, general dilatation of the base of the aorta, or aneurismal dilatations immediately above the aortic segments, are the conditions which produce it.

It is said that 'relative' incompetence of the pulmonary artery may be produced in the same manner as relative incompetence of the aortic orifice (by increased pulmonary tension, etc.); but no case of this description has come under my own observation.

Pathological physiology.—Now all lesions which impair the efficiency of the valves, *i.e.* all valvular lesions which produce incompetence or stenosis, or a combination of these conditions, interfere with the steady onward passage of the blood in the normal direction, and tend to produce anæmia in front (arterial anæmia) and congestion behind (venous plethora). I say *tend* to produce, for, as we have previously seen, compensatory changes in the heart are almost invariably established, which, for a time at least, restore the balance of the circulation.

The effects, in short, of all lesions which impair the efficiency of the valves, are partly mechanical and partly vital, and vary with:—

1. *The valve which is affected, and the exact nature of the lesion.*—The effects which are produced for instance, by mitral stenosis, both upon the heart and upon the peripheral venous and arterial systems, and hence upon distant organs, are very different from those which result from aortic regurgitation.

2. *The suddenness of the lesion.*—When a valvular lesion is quickly established, as, for example, when an aortic valve-cusp is ruptured, a sudden strain is thrown upon the cavity of the heart which is placed immediately behind the lesion. In consequence of the sudden increase of the blood-pressure, dilatation of the cavity is (apt to be) produced, and hypertrophy is only subsequently established.¹

When, on the contrary, a valvular lesion is slowly and gradually established, the blood-pressure behind the lesion is very gradually increased. The muscular wall of that cavity of the heart which is situated immediately behind the lesion is stimulated by the gradually increasing blood-pressure; and provided that the vitality of this organism as a whole, and of the heart in particular is good, the increased stimulation is attended (in the case of obstructive lesions at all events) with the production of a slowly developing hypertrophy, which for a time keeps pace with the valvular lesion and counterbalances the defect. In the case of obstructive lesions, then, which are slowly and gradually established, hypertrophy of the cardiac wall behind the lesion is gradually produced, and it is only subsequently that dilatation is set up. This statement applies more especially to aortic stenosis, for in the case of the mitral valve the strain on the thin walled auricle is probably from the first attended with some dilatation. We shall afterwards see that in the case of regurgitant lesions the tendency to the production of dilatation,

¹ In the lower animals artificial lesions of the valves are immediately compensated by the reserve force which the heart possesses. In the human subject lesions of this description seldom occur, except in the case of valves previously weakened by disease; and in such cases very serious derangement of the circulation is, as a rule, immediately set up. In other words, the reserve force possessed by the human heart, is seldom sufficient to *completely* and *immediately* remedy such a sudden and severe lesion.

in the earlier stages, is even greater than in cases of stenosis; for the increased blood-pressure during diastole, while it undoubtedly stimulates the flaccid muscular wall to contract more rapidly than is natural, tends also to produce dilatation. In regurgitant lesions, then, which are slowly and gradually established, some dilatation in addition to hypertrophy is usually produced, even in the early stages of the case.

3. *The extent of the lesion.*

4. *The general vitality of the individual and the special vitality of the heart in particular.*—If the general tone of the organism is below *par*, and more particularly if the special vitality of the cardiac muscle is interfered with, either as the result of defective blood-supply (disease of the coronary arteries), defective nerve supply, or structural defects, such as fatty or fibroid changes, the cardiac muscle does not respond to the increased stimulation, there is little or no compensation, and dilatation and not hypertrophy is produced.

The nett result, then, as regards the mechanical derangement of the circulation, which is produced by any valvular lesion, is determined by the suddenness and extent of the lesion on the one hand, and by the amount of the compensatory changes in the heart on the other.

When we come to measure the results *on the organism as a whole*, we have to take into account the condition of the peripheral organs (*i.e.* the organs peripheral to the heart—the centre of the circulation). The same amount of mechanical derangement of the circulation will produce very different effects, for example, in the case of two individuals, one of whom is healthy and the other in a state of disease. In other words, the resisting power to the mechanical derangement of the circulation possessed by the peripheral tissues and organs, more particularly the condition of the digestive apparatus, the kidneys, and the nerve centres, is a most important factor in determining this result.

With these preliminary remarks we will now consider the lesions of the different valves in detail.

MITRAL INCOMPETENCE.

Definition.—Mitral incompetence includes all those conditions, which interfere with the perfect closure of the mitral valve apparatus, and allow a regurgitant blood current to pass from the cavity of the left ventricle to that of the left auricle, during the ventricular contraction or systole.

Ætiology and Pathology.—Mitral regurgitation is a very common condition; it is met with at all ages,¹ and in a variety of affections, and may result from a lesion of any part of the mitral valve apparatus (sphincter muscle, basal ring, valve segments, chordæ tendineæ, or papillary muscles); but in order that the exact manner of its production may be clearly understood, I must direct attention, more minutely than I have hitherto done, to the construction of the mitral valve apparatus, and to the manner in which its closure is effected.

The function of the mitral valve apparatus is, of course, to close the mitral orifice and to prevent any regurgitation from the cavity of the left ventricle to that of the left auricle, during the ventricular systole. This closure is effected:—

(1) *By the narrowing of the mitral orifice, which results from the ventricular contraction.*

The mitral orifice is surrounded in its posterior two-thirds by the muscular fibres of the left ventricle; while the anterior third, which is fibrous, is formed by the fibrous continuation of the two posterior aortic sinuses to which the great anterior flap of the mitral valve is attached. (See figs. 177 and 178.) Now during the ventricular systole the muscular fibres surrounding the mitral orifice, of course, contract; and (as Macalister has shown in the admirable lecture in which he has described the most important observations of Ludwig and Hesse on the form and mechanism of the heart) ‘when systole is complete, the area of the orifices is not much more than half of what it is in diastole.’ (See fig. 179.)

¹ Regurgitation due to organic changes in the valve segments, which is generally due to rheumatic endocarditis, is more common in young than in old people.



FIG. 177.—Portion of the aorta and wall of the left ventricle with one entire segment and two half segments of the aortic valve, and the right or anterior segment of the mitral valve; *a, b, c*, sinuses of Valsalva opposite the segments; in *a*, and *b*, the apertures of the coronary arteries are seen; *d, d'*, the inner surface of the wall of the ventricle; *1, 2*, curved attached border of the segments; *3*, the middle of the free border (corpus Arantii); *e, e'*, the base of the anterior segment of the mitral valve; *f*, its apex; between *e*, and *e'*, and *f*, the attachment of the branched chordae tendineae to the margin and outer surface of the valve segment; *g*, right, *h*, left principal papillary muscle: the cut chordae tendineae are those which belong to the posterior segment and the small or intermediate segments.—(After Quain.)

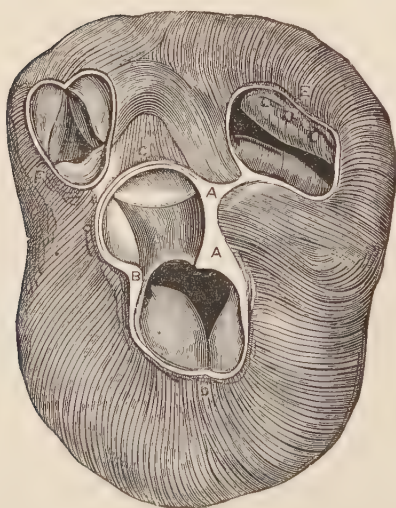


FIG. 178.—Calf's heart boiled, showing the aortic (C) and mitral (D) orifices thrown into one by the removal of the mitral valve, the lower A being the central fibrocartilage, E the tricuspid orifice, and F the orifice of the pulmonary artery.—(After Sibson.)



FIG. 179.—*Base of the ventricles in systole according to Ludwig; two-thirds natural size.—(After Macalister.)*

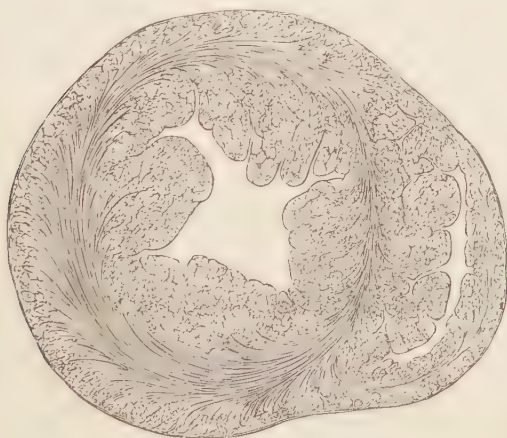


FIG. 180.—*Cross section of the ventricles in systole.—(After Macalister.)*

(2) *By the accurate apposition of the mitral segments.*—There are two mitral segments. The *anterior* flap, which is simple, and when closed is shaped like a three quarters

moon, 'is attached on the one hand to the junction of the left ventricle to the left auricle, and on the other to the roots of the right and left posterior flaps of the aortic valve. This attachment of the mitral to the aortic valve is effected through the fibrous structure that extends from the base of one valve to the base of the other, and by the central fibro-cartilage of the heart, which forms a triple bond of connection, that ties the mitral, the aortic, and the tricuspid valves to each other.' 'The posterior flap is compound, and when closed, is shaped like a quarter or crescent-shaped moon. The compound posterior flap is usually made up of one central and two lateral sub-segments, the latter being sometimes subdivided. These sub-segments adapt themselves so to each other, that the concavity of the crescentic border of the posterior compound flap is preserved entire; for it would have been impossible, by means of one simple fold of membrane, to fill up without a break the whole of the crescentic border.' . . . 'When the mitral valve is shut, the anterior flap of the valve presents a convex edge, shaped like a horse-shoe, which falls back upon, and fits like a lid into, the posterior flap of the valve, which flap, being crescentic in shape, presents a concave edge. Each flap adapts itself to the other by a notched lip, made up of small hemispherical eminences. The eminences of one lip fill up the notches of the other lip. These eminences thus seen on the auricular surface of the valve, are cells when seen on its ventricular surface, and as these cells are distended with blood when the ventricle contracts, and are exactly maintained in their places by the tendinous cords and papillary muscles, the distended cells or eminences at the opposite lips of the valve adapt themselves to and press against each other during the systole, so as to shut the valve.'¹ (See figs. 177, 181, 182, and 183, in which the relationship of the mitral valve segments is clearly shown.)

(3) *Further, the segments of the valve are maintained in position by means of the chordæ tendineæ and papillary muscles.*—During the ventricular contraction shortening of the pa-

¹ Sibson on the Form and Position of the Heart. *Russell Reynold's System of Medicine*, vol. iv. pp. 50, 51.



FIG. 181. Section through the aorta, left auricle, one of the aortic segments and the anterior cusp of the mitral valve, of a child three weeks old. (Magnified about 10 diameters.) The aorta has become detached from the auricle in the process of mounting.

a, inner, *b*, middle, and *c*, outer coats of the aorta; *d*, sinus of Valsalva; *e*, segment of aortic valve; *f*, *f'*, fat cells; *g*, *g'*, cellular tissue between the aorta and left auricle, which have become detached in the process of mounting; *h*, endocardium lining the left auricle; *i*, connective tissue; *m*, *m'*, *m''*, muscular fibres of the wall of the left auricle; *n*, anterior segment of the mitral valve; *v*, placed in a sinus or depression beneath the aortic valve cusp.

FIG. 182.—Section through the left auricle and left ventricle of a child three weeks old showing the attachment of the posterior segment of the mitral valve. (Magnified about 10 diameters.)

The posterior segment of the mitral valve has become curled up in the process of mounting; the relative thicknesses of the endocardium lining the auricle and ventricle respectively are well seen.

- a*, epithelial covering, and
- b*, connective tissue layer of the endocardium of the left auricle;
- c*, sub-endocardial connective tissue
- d, d*, muscular fibres of the left auricle;
- e*, connective tissue of the outer wall of the left auricle and of the pericardium;
- f*, surface of the pericardium covering the left auricle;
- g*, coronary vein transversely divided;
- h*, pad of fat between the auricle and ventricle;
- i*, pericardium covering the left ventricle;
- l*, large vein in the pericardium
- k, k*, muscular fibres of the left ventricle;
- n*, the turned up extremity of the posterior segment of the mitral valve;
- n'*, the point of attachment of a chorda tendinea to the mitral valve.

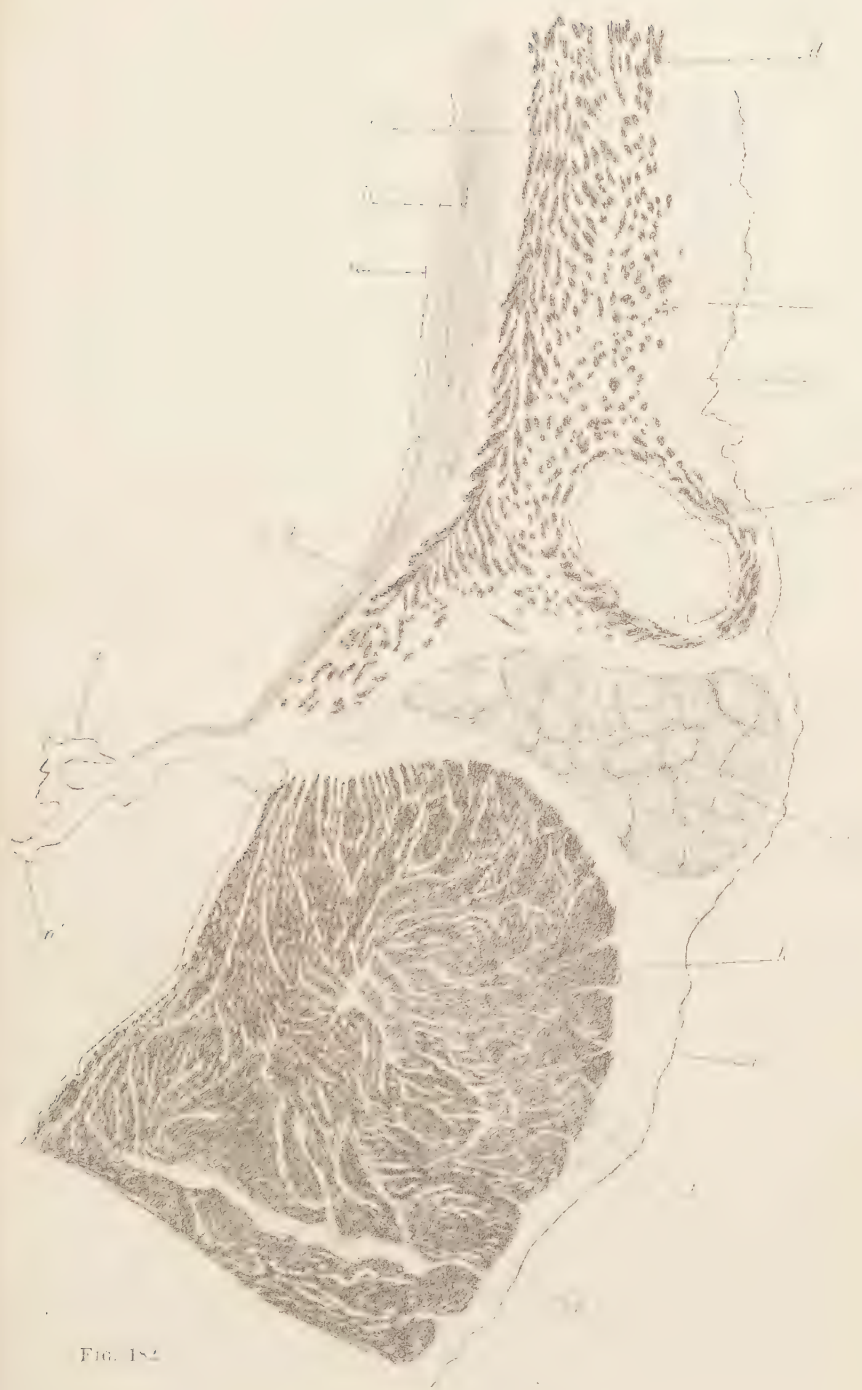


FIG. 182

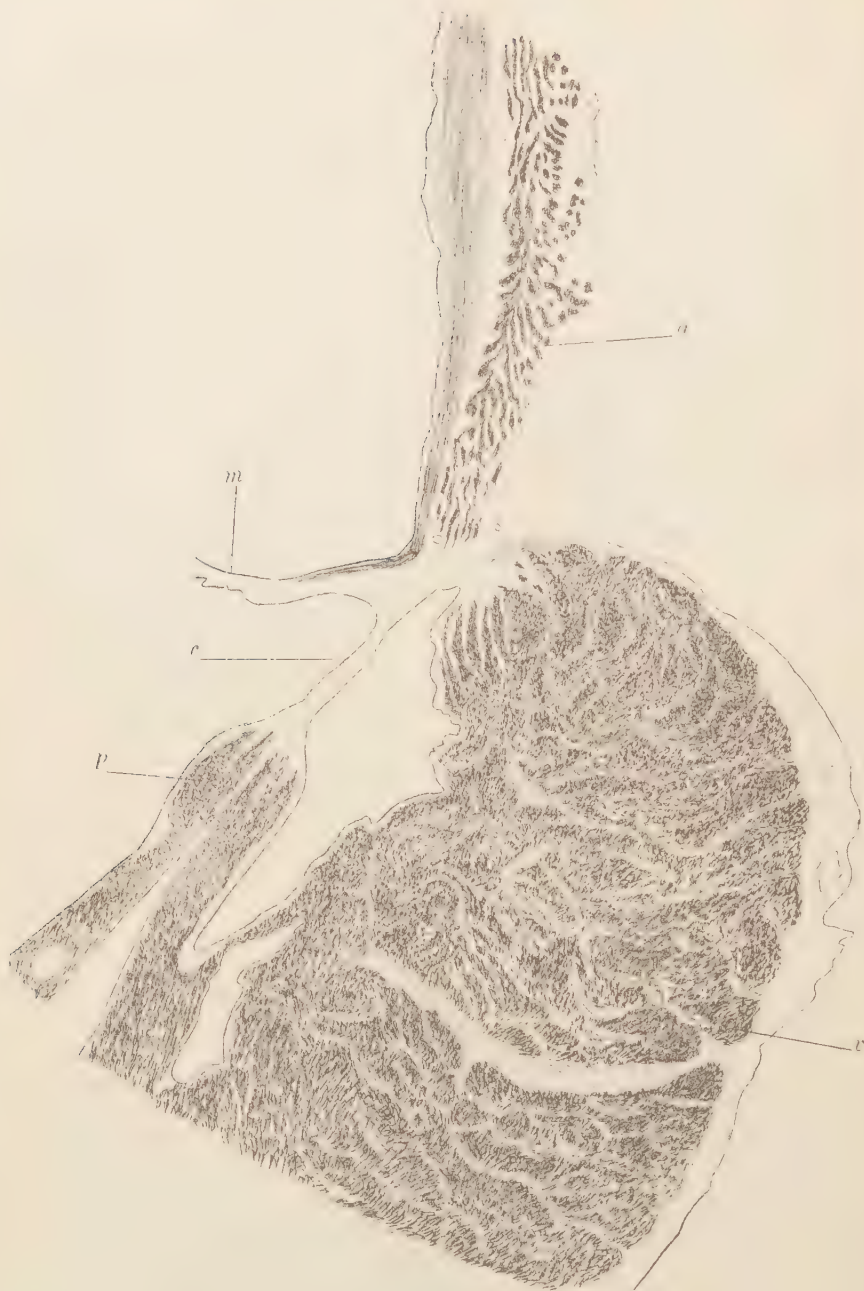


FIG. 183.—Section through the left auricle and left ventricle of a child three weeks old, showing a papillary muscle and its chorda in situ. Magnified about 10 diameters. (The pericardium and connective tissue on the outer surface of the left auricle have become detached in the process of mounting.)

p, papillary muscle; *c*, chorda tendinea; *m*, posterior segment of the mitral valve; *a*, wall of the left auricle; *v*, wall of the left ventricle.

pillary muscles occurs, and in consequence of this shortening the various parts are maintained 'at the same levels in which they lay in diastole.'¹ In this way retroversion of the flaps into the auricle during the ventricular systole is prevented.

Regurgitation or incompetence may, therefore, be due to the following pathological causes:—

1. *Defective muscular closure of the valve.*—This condition, which I term *muscular incompetence*, and which is very often combined with dilatation of the basal ring (relative incompetence) is a common cause of mitral regurgitation. Defective muscular closure is produced by anything which impairs the muscular tone of the left ventricle as a whole, and of the muscular fibres which surround the mitral orifice (the mitral sphincter, as I term it) in particular. In practice we find this form of mitral regurgitation associated with:

(a) Chlorosis, pernicious anæmia, and other anæmic conditions, in which the muscular fibres of the left ventricle are limp and fatty. It must, however, be remembered, that mitral regurgitation is often not produced by the fatty degeneration of the heart which occurs in old people—the fatty degeneration, for example, which is seen in connection with disease of the coronary arteries and general atheroma. This apparent exception can, I think, in some cases, be explained by supposing that the basal tendinous ring, which surrounds the mitral orifice, is thickened or even calcareous; and that in consequence of the altered condition of the basal ring there is stenosis of the orifice, or, at all events it is so rigid that dilatation (relative incompetence) cannot occur.

(b) Myocarditis, more especially those cases in which the mitral sphincter is involved. I have already alluded to this as a probable cause of the mitral regurgitation which is so common in acute rheumatism. (See page 374.)

(c) The degenerative changes in the cardiac muscle which are met with in the later stages of typhus, typhoid, and the other continued fevers.

¹ *Lecture on the Form and Mechanism of the Heart*, by Dr Donald Macalister, *British Medical Journal*, Oct. 28, 1882, p. 825.

(d) Fibroid degeneration of the left ventricle. This is not a frequent cause of mitral incompetence ; *firstly*, because fibroid degeneration of the left ventricle is not very common ; and, *secondly*, because in many cases in which the left ventricle is affected with fibroid degeneration, the conditions for the production of mitral regurgitation are not established. If, for instance, the mitral sphincter remains sound, or, if the basal ring is narrowed, as it often is in consequence of the endocarditic changes, which are so frequently associated with fibroid degeneration of the wall of the heart, the valve flaps may be quite competent to close the orifice.

(e) Grave's Disease (Exophthalmic Goitre).—In many of these cases, mitral regurgitation, the result of muscular and relative incompetence, occurs.

Stenosis is rarely combined with 'muscular' and 'relative' incompetence. On examining the heart in cases of this description, therefore, after death, the valve may appear to be perfectly natural. We can only¹ judge of the competence of the mitral valve (1) by accurate measurement of the orifice, and (2) by careful microscopical examination of its sphincter muscle. And in judging of its competence by measurement, it is important to remember that *post-mortem* contraction of the left ventricle diminishes the size of the orifice, just as *ante-mortem* contraction does.

2. *Relative Incompetence*.—This condition is, as I have mentioned above, generally combined with 'muscular' incompetence. It occurs more particularly in those cases in which the cavity of the left ventricle is dilated and its muscular wall degenerated. In the later stages of aortic regurgitation, mitral regurgitation due to a combined condition of 'relative' and 'muscular' incompetence, is of common occurrence.² High arterial tension, when combined with impaired tonicity, fatty degeneration, etc., of the left ventricle, also gives rise to it.

¹ The water test is useless in those cases in which the left ventricle is relaxed and flaccid. It can only be properly applied in those cases in which the heart is in a condition of rigor, and the left ventricle firmly contracted.

² The mitral regurgitation, which is sometimes associated with increased arterial tension, is in other cases due to chronic endocarditis, set up by the violent strain to which the segments are subjected.

3. *Structural changes in the valve segments, chordæ tendineæ, and papillary muscles.*—In cases of this description the incompetence is very generally combined with more or less stenosis of the orifice.¹ The incompetence may be due to the following conditions:—

(a) *Acute Endocarditis.*—This cause of mitral regurgitation has already been fully considered. (See page 371, *et seq.*) It will be remembered that, during the acute stage, the incompetence is generally *pure*, *i.e.* unassociated with stenosis.

(b) *Chronic Endocarditis.*—The thickening and retraction of the valve segments, or retraction of the chordæ tendineæ, prevent the accurate apposition of the valve flaps, and regurgitation consequently occurs. Regurgitation may result from elongation and stretching of the chordæ tendineæ, which have become softened by the inflammatory process. The mitral flaps are consequently no longer held in accurate apposition, but are forced too far back during the ventricular systole.

(c) *Rupture of the chordæ tendineæ.*—This is fortunately a comparatively rare condition. The regurgitation which results is very free, and the symptoms proportionately severe. More than one cord may give way. Rupture of a perfectly healthy tendinous cord probably never occurs. In the cases which have come under my own observation in the *post-mortem* room (such as that represented in figs. 169 and 170), there have been well-marked appearances of endocarditis.

(d) *Fatty and fibroid changes in the papillary muscles.*—In fatty conditions of the left ventricle, notably in progressive pernicious anæmia, the papillary muscles are implicated. They are, also, frequently the seat of fibroid changes. In chronic endocarditis, for example, the tips of the papillary muscles to which the thickened tendinous cords are attached, are very frequently involved, and in some cases the whole muscular fibre of one or more of the papillary muscles is

¹ Incompetence due to organic changes in the valve segments is frequently combined with regurgitation due to 'muscular' or 'relative' incompetence. This is more particularly the case in the terminal stages of the disease, when the muscular fibre of the left ventricle has become degenerated.

replaced by fibrous tissue. In both of these conditions, but more especially in the former (for in fibroid degeneration of the papillary muscles, the chordæ tendineæ are often retracted) the mitral segments are not held accurately in position during the ventricular contraction, but are floated too far back into the orifice ; regurgitation consequently results.

(*e*) Atheroma.—The anterior segment of the mitral valve is occasionally affected with atheroma, though much more rarely than the aortic cusps. In many cases the atheromatous deposits do not interfere with the competence of the valve ; in others (and in these cases chronic endocarditis is usually present) the valve segments become thickened, rigid, and incompetent.

Pathological Physiology.—The *first* effect of mitral regurgitation is, of course, to allow some of the blood, which ought to be pumped into the aorta during the ventricular systole, to pass back into the left auricle ; in other words, to produce more or less engorgement behind, and more or less anæmia in front of the cavity of the left ventricle.

The *second* effect is to produce a series of changes in the heart itself, the other parts of the circulatory system, and in the peripheral organs (*i.e.* the organs peripheral to the heart—the centre of the circulation).

Some of these changes are, as I have so repeatedly pointed out, eminently salutary and compensatory in character ; others are as decidedly prejudicial.

The extent of the vascular engorgement behind, and of the anæmia in front, is equal, as we have previously seen, to the amount of the regurgitation *less* the degree of compensation ; while the extent of the tissue changes is determined by :—

(1) The amount of the vascular derangement (*i.e.* of the engorgement behind and the anæmia in front).

(2) The vitality or resisting power of the tissues as a whole, and of certain special organs, such as the liver, stomach, kidneys, and nerve centres, in particular.

But since it is of the utmost importance, both for the due comprehension of the symptoms and for the purposes of

intelligent prognosis and treatment, to understand clearly the exact nature of these changes, we must consider them in further detail.

Effect on the left auricle.—The regurgitant current passes into the left auricle during its diastole, *i.e.* when its walls are flaccid, and meets the blood current which is being poured into its cavity by the pulmonary veins. The cavity of the left auricle is, therefore, more forcibly and more rapidly dilated than in health; its muscular wall is more frequently and more powerfully stimulated; it contracts more frequently, and tends to become hypertrophied. The hypertrophy is seldom great; for, in the *first* place, the muscular wall of the auricle is very thin, and is much more easily dilated than hypertrophied; and, in the *second* place, the primary lesion, which produces the regurgitation, is very frequently a degenerated or debilitated condition of the ventricular muscle. In cases of this description (*i.e.* cases of muscular and relative incompetence) the auricular muscle is very generally in a similar condition (degenerated or debilitated) and incapable, therefore, of much hypertrophy.

In addition, the fibrous layer of the endocardium lining the auricle usually becomes thickened. The usual result is, therefore, that the cavity of the left auricle becomes more or less dilated, its muscular wall more or less hypertrophied, and the fibrous layer of the endocardium thickened.

Now all of these changes are under certain circumstances salutary. In those cases, for instance, in which the auricle becomes just sufficiently dilated to accommodate the quantity of blood, which passes back at each ventricular systole, and at the same time to receive the normal quantity of blood from the lungs, and in which the muscular wall of the auricle becomes sufficiently hypertrophied to expel *all* the auricular contents (both the blood passing into its cavity from the lungs and from the left ventricle), there is little or no congestion behind the cavity of the left auricle, and the lesion is practically compensated. As a matter of fact, such perfect compensation is very rarely met with, except in those cases in which the regurgitation is small in amount, and in which

the vitality of the cardiac muscle is extremely good. Examples of such perfect compensation do however occur, more particularly in cases of mitral regurgitation in which the incompetence is caused by organic changes in the valve segments, and in which the patient is young and healthy. Perfect compensation of this description is seldom if ever seen in 'muscular' or 'relative' incompetence or in *free* regurgitation, the result of organic changes in the valve segments.

Effects on the Pulmonary Circulation.—In almost all cases of mitral regurgitation the pulmonary circulation is obstructed. In the cases of perfect compensation, to which I have just referred, the obstruction may be so slight as to be practically ignored. In the great majority of cases, it is much more considerable. The free passage of the blood from the pulmonary veins into the cavity of the left auricle is interfered with, more particularly during the period of the ventricular systole. The tension of the blood in the whole pulmonary circuit (the pulmonary veins, pulmonary capillaries, and pulmonary artery) is consequently increased. Dilatation of the pulmonary vessels, more especially of the pulmonary capillaries, and other secondary changes in the pulmonary tissue, to which I shall presently refer more in detail, occur.

The obstruction in the lungs during the ventricular systole, throws an increased strain on the right ventricle of the heart, in consequence of which its muscular fibres are over-stimulated and (when healthy) become hypertrophied.

The dilatation of the pulmonary capillaries is, in the earlier stages at least, seldom so great as in mitral stenosis, for in mitral regurgitation the increased blood pressure in the pulmonary circuit is not so continuous as it is in stenosis of the mitral orifice. In cases of mitral regurgitation (I am speaking of comparatively *pure* cases in which there is little or no stenosis combined with the incompetence), as soon as the ventricular systole ceases, the mitral valve is of course thrown open, and the blood pressure in the pulmonary circuit is suddenly lowered. In mitral stenosis, on the contrary, the passage of the blood from the left auricle to the left ventricle, through the narrowed orifice, is a gradual process; and the fall of the

blood pressure in the pulmonary circuit is much more slowly brought about.

It must also be remembered that in mitral stenosis, and in those cases of mitral regurgitation in which the muscular wall of the left ventricle is degenerated, the suction action of the left ventricle is much less perfect than normal, and the passage of the blood through the lungs is, from this cause, rendered more difficult than in health. In the healthy condition of things, 'the force that overcomes the resistance of the lung capillaries is not entirely a *vis a tergo*. The left ventricle does some of the work of the right. To produce this elastic straining in its walls, of course requires a greater muscular effort in the contraction; but the energy is not lost or wasted. When diastole begins, it is restored—for the benefit of the pulmonary circulation.'¹ In those cases, therefore, of mitral regurgitation in which the ventricular muscle is debilitated or degenerated, an additional strain is thrown upon the right ventricle in consequence of the diminished suction power of the left heart; *vice versâ* in those cases in which the muscular wall of the left ventricle is healthy and hypertrophied, the suction power of the left heart may be actually increased, and the flow of blood through the lungs during the ventricular diastole facilitated.

Changes in the right heart.—As I have just explained, the obstruction to the passage of the blood through the lungs, which results from mitral incompetence, throws an increased strain on the right ventricle of the heart. In some cases, more especially in young healthy patients, and in cases in which the mitral lesion has resulted from organic changes in the valve segments, the right ventricle responds well to the increased stimulation, and becomes hypertrophied. This is another eminently favourable and compensatory result. Unfortunately, in many cases (as, for instance, in all those conditions in which the muscular tissue of the right ventricle is debilitated and diseased), the response is imperfect, and dilatation,

¹ *Lecture on the Form and Mechanism of the Heart*, by Dr Donald Macalister, —*British Medical Journal*, Oct. 28, 1882, p. 824.

or a combined condition of dilatation and hypertrophy results. In proportion as the dilatation exceeds the hypertrophy, so is the result injurious. When the muscular wall of the right ventricle is debilitated, and when the cavity of the right ventricle is dilated, tricuspid regurgitation, as the result of 'muscular' and 'relative' incompetence is established,¹ and the last barrier which protects the superior and inferior venæ cavæ and the veins of the heart itself, from the baneful effects of backward pressure, is removed.

With the occurrence of tricuspid regurgitation the cavity of the right auricle becomes dilated, the fibrous layer of its endocardial coat thickened, and in some cases its muscular wall hypertrophied. It is seldom, however, that the hypertrophy is sufficiently great to be of any practical effect.

Effect on the left ventricle.—Before considering the results of congestion of the systemic venous circulation, which follow next in order, as we proceed backwards from the mitral valve to the periphery, it will perhaps be well to direct attention to the alterations which are produced in the cavity of the left ventricle, and to the conditions which result therefrom.

As soon as the systole terminates, the elastic recoil, so to speak, of the left ventricle occurs; the mitral valve is thrown open, and a larger quantity of blood than normal passes with unusual force from the cavity of the left auricle into that of the left ventricle. The cavity of the left ventricle is, therefore, more quickly distended, and its muscular wall more powerfully stimulated than in health. The increased stimulation, and the effort which is required to expel the unusually large quantity of blood, result in the production of hypertrophy, in those cases in which the vitality of the muscular tissue is good. In other cases (*i.e.* when the vitality of the cardiac muscle is impaired) dilatation, rather than hypertrophy, occurs.

¹ Muscular and relative incompetence are much more readily established at the tricuspid than at the mitral orifice. This is, of course, just what we would expect, —the muscular wall of the left ventricle being so much more powerful than that of the right. The condition of the cardiac muscle—its capacity of becoming hypertrophied—is, therefore, a most important factor in determining the progress of mitral regurgitation, as I shall afterwards point out more in detail.

In consequence of the hypertrophy, the left ventricle is enabled to expel a larger quantity of blood than it otherwise could, into the aorta and arterial system. In this respect, therefore, the hypertrophy is beneficial and compensatory. On the other hand, the powerful contraction of the left ventricle drives an increased quantity of blood through the incompetent mitral orifice. In this respect, therefore, the hypertrophy is bad.

In cases of mitral regurgitation, the hypertrophy is seldom great, and is usually combined with more or less dilatation of the ventricular cavity. In judging of the amount of hypertrophy after death, it is important to note the condition of the muscular fibre, as well as the thickness of the wall of the ventricle and the weight of the heart. Increased thickness of the wall of the ventricle may, of course, be due to other conditions than increase of its muscular fibres (such, for instance, as fibroid degeneration, fatty deposits, and thickening of the pericardium). In many of the cases of mitral regurgitation, in which the muscular fibre becomes hypertrophied, degenerative changes, such as fibroid and fatty degeneration, subsequently occur, in consequence of the venous hyperæmia in the cardiac walls, and the deficient supply of healthy arterial blood resulting from an advanced mitral lesion.

It must also be remembered that the hypertrophy of the left ventricle may be due to extra-cardiac causes, such as cirrhosis of the kidney, atheroma of the arterial system, etc. It is also probable that in advanced stages of mitral disease, the venous engorgement and capillary congestion, by impeding the flow from the arterial to the venous system, throw an increased strain upon the left heart, and so produce hypertrophy of the walls of the left ventricle.

Effects on the Systemic Venous Circulation and Peripheral Organs.—With the occurrence of dilatation of the right heart, and more particularly after tricuspid incompetence has become fully established, the systemic venous circulation, which is usually from the first more or less interfered with, becomes seriously embarrassed; the watery parts of the blood tend to escape into the subcutaneous cellular tissue and great

lymphatic sacs (peritoneum, pleuræ, pericardium), and the secondary alterations, which the venous congestion has been slowly producing in the walls of the heart and in the peripheral organs, become much more prominent.

These alterations, which are partly also due to a deficient supply of healthy arterial blood are of the greatest practical importance, and must be considered in détail.

Alterations in the Lungs.—In consequence of the increased tension of the blood in the pulmonary circuit, the pulmonary capillaries become dilated and encroach more upon the air space (the interior of the air vesicles) than in health. These thin walled and dilated vessels are easily ruptured by any sudden increase of the blood pressure, and hæmoptysis and pulmonary apoplexy are therefore apt to occur.¹

The venous congestion of the bronchial veins, which results from engorgement of the systemic venous circulation leads to a similar change (dilatation) in the nutrient vessels of the lungs and bronchi; catarrh of the air vesicles and bronchial mucous membrane is apt to arise, and the condition which has been termed brown induration of the lungs is established. Œdema of the lungs, and low forms of pneumonia, are also very apt to occur.

Liver.—The branches of the hepatic vein become dilated, and the liver is at first increased in size. After a time the liver cells, more especially those in the centre of the lobules, *i.e.* in the zone of distribution of the hepatic vein, become atrophied and fatty; the corpuscular elements of the blood (seen after death in the form of pigment granules and leucocytes) escape from the dilated and congested capillaries into the spaces between the liver cells. Ultimately the organ becomes tougher and (sometimes) smaller than normal, and after death is found to present the nutmeg and cirrhotic appearance.

Spleen.—The spleen becomes engorged and enlarged; and its fibrous stroma increased. After death it is found to be large, tough, and of a dark purple colour.

¹ Hæmoptysis and pulmonary apoplexy seldom occur in the early stages of mitral regurgitation. In this respect mitral incompetence differs from mitral stenosis in which spitting of blood is often an early symptom.

Stomach and Intestines.—The venous radicles and capillaries become enormously enlarged, the secreting structures are apt to become atrophied, and a catarrhal condition of the mucous membrane established.

Kidneys.—Chronic congestion of the kidneys is attended with a scanty flow of high-coloured urine, which deposits a copious sediment of urates and often contains albumen. After death the kidney is found to be firm, tough, and dark-coloured. A form of cirrhosis, in which the organ may be of normal size or larger than natural, is ultimately established.

Pelvic Viscera.—Chronic venous congestion of the pelvic viscera leads in the female to leucorrhœa and derangements of menstruation.

Nerve Centres.—The venous radicles of the nerve centres share in the general congestion; the nutrition of the nerve elements is imperfectly carried on, and an œdematous condition, with effusion into the ventricular system, ultimately occurs. The impaired nutrition of the trophic and other nerve centres, reacts upon all the peripheral organs and tissues, and the vitality and resisting power of the whole organism, including the heart itself, become seriously impaired.

Heart.—It is extremely important to remember that the return current of blood through the cardiac veins is also obstructed; the venous radicles and capillaries throughout the heart become engorged; their contents tend to escape into the lymphatic spaces between the muscular fibres; and the nutrition of the muscular fibres is interfered with. After death the cardiac walls are usually found to be tougher and harder than natural. In those cases in which the muscular fibres are fatty, the cardiac wall may be soft and pale.

In figure 184 I have attempted to represent in a diagrammatic manner the effects which are produced in the peripheral organs by the congestion of the venous system.

Alterations in the Composition of the Blood.—In advanced stages of mitral disease the composition of the blood is profoundly altered, and, in consequence of this alteration, the nutrition of the tissues and organs, including, of course, the heart itself, is very imperfectly carried out. The altered

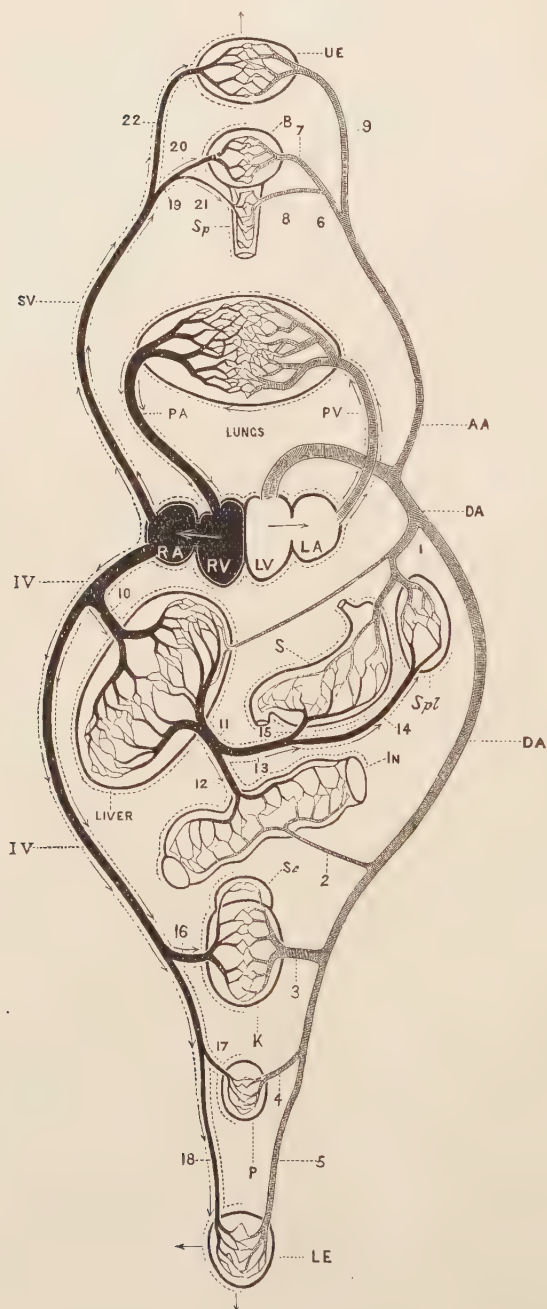


FIG. 184.

composition of the blood is due to the following conditions:—

1. *Imperfect formation and elaboration.*—The appetite is impaired, the amount of food ingested is less than normal. The functional activity of the stomach and other organs, concerned in the manufacture of the blood, is seriously damaged in consequence of the deficient supply of healthy arterial blood, and of the venous congestion and its results, which have been already described. The impaired functional activity of the nerve centres reacts, of course, upon the stomach and other chylopoietic viscera, and adds further difficulties to the digestive process; 2. *Imperfect elimination of waste products.*—The functional activity of the liver, kidneys, intestines, and skin is, as we have already seen, seriously interfered with and the blood becomes loaded with effete products; 3. *Imperfect aeration.*—In consequence of the imperfect aeration of the blood in the lungs, which is one of the most marked features in advanced cases of mitral disease, the blood contains a larger quantity of carbonic acid, and less oxygen than usual; 4. *Loss of water and albumen,* which occurs in consequence of the dropsical effusions, and of the escape of albumen in the urine.

Clinical History.—Having previously described in detail the clinical history of mitral regurgitation, due to acute endocarditis, I will limit the following remarks to the chronic forms of the disease. And in considering the symptomatology of chronic mitral incompetence, it is essential to make a distinction between (1) mitral regurgitation due to organic changes in the valve segments, in which the mitral lesion may be said to be the primary cause of all the subsequent symptoms, and (2) mitral regurgitation due to other conditions, such, for instance, as the fatty degeneration and muscular relaxation of chlorosis and progressive pernicious

Description of Fig. 184.

Diagrammatic representation of the effects of a mitral lesion upon the venous circulation. The numbers and letters are the same as in fig 3. (See description page 5.) The arrows indicate the direction of the backward current.

anæmia, in which the symptoms are, for the most part, due to the general condition—in chlorosis and progressive anæmia—the examples which I have taken—to the anæmic condition.

It is important also to remember that, in the first group of cases, associated lesions, such as aortic valvular disease, affections of the kidney, etc., are common, and complicate the clinical picture of the case.

Let us consider, then, in the first place, the clinical history of the chronic mitral incompetence, which is due to organic changes in the valve segments. The symptoms of this condition, which usually results from endocarditis or atheroma, and which is generally associated with some constriction of the valvular orifice, may follow the symptoms of an acute or subacute attack of endocarditis, or may be very slowly and gradually developed in a person who has previously enjoyed good health.

The symptoms vary considerably in different cases, but are, for the most part, due to mechanical derangement of the circulation in the peripheral organs (*i.e.* in the organs peripheral to the right and left hearts). Subjective cardiac sensations are seldom prominent. In advanced stages of the case, *i.e.* when the dilated cardiac cavities are imperfectly emptied, clots sometimes form within the heart, and embolic symptoms due to the plugging of distant vessels occasionally arise.

In some cases, the lesion is so slight and so perfectly compensated, that although the presence of a permanent apex systolic murmur indicates to the medical attendant the presence of mitral regurgitation, there may be no symptoms experienced by the patient. The lesion may remain in this latent, inactive condition, for years. In some cases, it neither interferes with the comfort of the patient, nor shortens life.

In others, and these are the majority, there is shortness of breath on any extra exertion, while dropsy and the other symptoms and signs of a progressive mitral lesion are ultimately developed, and lead to a fatal termination. The occurrence of symptoms, after a long period of latency or inactivity, is usually due either to a subsequent more active development of the valvular lesion as the result of chronic

endocarditis, atheroma, etc., or to the failure of compensation. Both of the conditions are particularly apt to arise between the ages of fifty and sixty, when the degenerative processes of old age and decay are beginning to be prominent.

In other cases, the symptoms gradually progress, it may be with periods of temporary compensation and intermission, from the time when the patient first comes under observation; and after a longer or a shorter period, which depends upon the extent of the lesion and the vitality of the individual, the case terminates in death.

In all of these cases, and more especially in the last, urgent cardiac symptoms may be suddenly developed, and may depend upon:—

- (1) The occurrence of acute endocarditis.
- (2) Rupture of the tendinous cords.
- (3) Acute pulmonary complications (such as acute bronchitis, pneumonia, œdema of the lungs, pleurisy with effusion, etc.) which suddenly add to the difficulties of the respiration and to the engorgement of the right heart and of the venous circulation.

The first symptoms, which patients affected with mitral regurgitation complain of, are generally due to engorgement of the pulmonary vessels, and are shortness of breath on exertion, going up stairs, up a hill, etc.; and slight cough. As the case advances, the shortness of breath becomes more marked, and the cough, which is sometimes short and dry, at others accompanied with more or less bronchial secretion, becomes more frequent and troublesome.

Palpitation, intermittent action of the heart, or a sinking, 'wanting,' sensation in the pit of the epigastrium are sometimes also experienced. Derangement of the digestive organs, loss of appetite, foul tongue, flatulence (which by displacing the diaphragm upwards adds to the mechanical difficulties of the lungs and heart) and constipation, or, more rarely, constipation alternating with diarrhœa, are common even at comparatively early stages of the case. A dingy colour of the skin, or even slight jaundice, and piles sometimes occur at this stage of the case. The patient feels depressed

and languid, and is not disposed for bodily exertion or mental work. A close observer can usually detect some blueness of the lips, ears, nose, fingers, or other peripheral parts; and distended congested vessels are often seen coursing over the cheeks and sides of the nose.

After a time the stage of dropsical effusions is reached, and all the symptoms become more prominent. The dropsy is usually first observed about the ankles, and only at night. As the case progresses it becomes permanent, and gradually extends upwards. The subcutaneous cellular tissue of the lower extremities, scrotum, and dependent parts of the back may become enormously swollen. Effusion also takes place into the internal cavities, more particularly the peritoneum, pleuræ, and pericardium. When the mitral lesion is complicated with organic renal disease, the dropsy may, of course, be general. When again cirrhosis of the liver is associated with mitral regurgitation, the dropsy into the peritoneal cavity is much more extensive than in ordinary cases of mitral disease.

The shortness of breath,¹ which has gradually increased with the advance of the cardiac lesion and with the occurrence of the pathological alterations in the lungs which have been previously described, may now be constant, and amount to orthopnœa. The derangement of the stomach, liver, and digestive organs becomes greater. Capillary hæmorrhages into the subcutaneous tissues, and purpuric eruptions are commonly observed in advanced stages of the case.

The nutrition of the body is seriously interfered with; the patient loses flesh; the number of red blood corpuscles is diminished, and notwithstanding the cyanotic condition of the periphery, lips, ears, nose, etc., a condition of general anæmia

¹ The shortness of breath, which is such a striking feature of mitral incompetence, may depend upon a variety of causes, amongst which the following are some of the chief:—

1. An increased quantity of blood in the lungs requiring oxydation, and a diminished quantity of air in the air cells. The diminished air space is, to some extent, due to the fact that the dilated pulmonary capillaries encroach upon the air vesicles, partly to the chronic catarrhal condition of the lungs and bronchi.

2. Secondary changes in the lungs and bronchi, such as bronchitis, emphysema, œdema, pulmonary apoplexy, etc.

3. Secondary changes in the pleuræ, such as hydrothorax, pleurisy.

is, to some extent, established. The urine is scanty in amount, high coloured, depositing a copious sediment of urates, and in many cases containing albumen. The functions of the nerve centres are seriously deranged; in some cases the patient is irritable and sleepless; in others drowsy. Erythema, erysipelas, or even gangrene of the skin of the swollen (dropsical) parts is apt to occur. Spitting of blood, the result of pulmonary apoplexy, is of common occurrence. The respiration becomes still more seriously embarrassed. Cheyne-Stokes' respiration may occur, and after a prolonged period of terrible suffering death takes place.

The clinical history of mitral regurgitation, due to muscular and relative incompetence, need not be specially described. In those cases in which the debilitated condition of the cardiac muscle depends upon a general pathological state, the clinical picture is a complicated one. In some cases, the symptoms, which depend upon the general (*i.e.* primary) condition, entirely overshadow those which are due to the mitral lesion. In others, the reverse holds good; in chlorosis and progressive pernicious anæmia, for instance, shortness of breath on exertion, and some œdema of the feet, are often prominent symptoms. Indeed, I have known more than one case of this description diagnosed, and that by no incompetent observer, as primary, organic, mitral disease. The distinctive features of these and other cases resembling primary, organic, mitral disease, will be presently detailed.

Physical signs.—The characteristic physical sign of mitral incompetence is a systolic murmur, having its point of differential maximum intensity at the left apex of the heart, and its direction of propagation outwards towards the left axilla. (See fig. 185.) The sound characters of the murmur and its extent of propagation vary very much in different cases, and depend upon the condition of:—

- (1) The left ventricle.
- (2) The mitral orifice.
- (3) The left auricle.

When the left ventricle is hypertrophied, and its muscular tissue healthy, when the mitral orifice is partly (though not

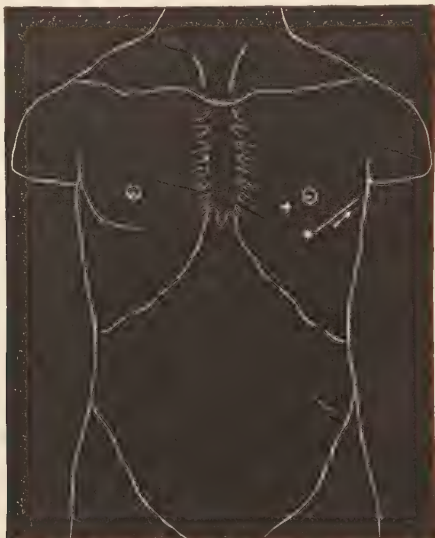


FIG. 185.—Outline figure showing point of differential maximum intensity (*) of the systolic mitral murmur (mitral regurgitation); and the direction in which it is propagated. The cross +, which is supposed to represent the normal position of the apex-beat, is placed a little too high.

extremely) constricted and its edges thickened, and when the cavity of the left auricle is dilated, the most favourable conditions for the production of a loud and extensively conducted murmur are present. In cases of this description, the murmur can be loudly heard in the back, beneath the inferior angle of the left scapula. *Vice versâ* when, as in pure muscular incompetence, the wall of the left ventricle is feeble, and when the annular ring is dilated rather than constricted, the murmur is soft and faint, and is badly propagated. In many cases of this description it cannot be heard beneath the inferior angle of the left scapula.

In some cases, an apex systolic murmur, indicative of mitral regurgitation, can of course be heard at the base of the heart as well as at the apex; but, for the reasons previously given, I do not agree with Naunyn, Balfour, and others, who believe that a systolic murmur, having its point of maximum

intensity in the second left interspace, at a point an inch or an inch and a half to the left of the sternum, and which is inaudible at the left apex, is indicative of mitral regurgitation.

In those cases of mitral disease in which the heart is acting very quickly, it may be difficult or impossible to detect a murmur. When, too, the left ventricle is contracting very feebly, the murmur is sometimes absent. In those cases of organic disease of the valve segments, in which there is stenosis as well as incompetence, the systolic apex murmur, indicative of regurgitation, may be preceded by a presystolic murmur indicative of stenosis.

In addition to the systolic murmur, which can be heard with the ear, a systolic thrill can sometimes be felt when the hand is placed over the apex of the heart. This sign is only, however, present in a minority of the cases of mitral incompetence, and even when present is not of very great practical importance, for in those cases in which a thrill is present, a murmur can almost invariably be heard. A thrill is in favour of the case being one of organic disease of the valve segments rather than 'muscular' or 'relative' incompetence.

In addition to the systolic apex murmur and the systolic thrill, which we may term the *primary* physical signs of mitral regurgitation, other physical signs, indicative of the secondary changes in the heart and circulation, and, therefore, of the greatest importance for the purposes of exact diagnosis, prognosis and treatment, are met with in most cases.

Amongst the *secondary* physical signs, as we may term them, the following are the chief:—

1. *Altered character of the radial pulse.*—The modifications in the radial pulse, which we meet with in mitral regurgitation, depend chiefly upon the extent of the lesion and the condition of the left auricle.

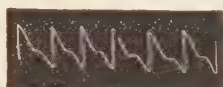
The *frequency* of the pulse is increased, for, in consequence of the leak at the mitral orifice, the left auricle is more quickly distended, and its wall is, therefore, more repeatedly stimulated than in health. The auricular contraction which is generated by this premature stimulation meets with no obstacle in

forcing the blood through the mitral orifice; the auricular contraction passes on to the left ventricle, and the frequency of the pulse is increased. The *volume* and *tension* of the pulse are diminished in consequence of the fact that some of the blood, which ought to be passed on to the aorta, passes backwards into the left auricle. The extent of the diminution depends upon the extent of the lesion and the condition of the left ventricle; when the regurgitation is free and when the left ventricle is failing, the pulse may be so small and feeble as to be almost imperceptible; *vice versâ* when the leak is slight and the left ventricle healthy, the volume and tension of the pulse are not much modified. *The rhythm of the pulse.*—In some cases the pulse is quite regular; in others, markedly irregular. These differences probably depend to a large extent upon the condition of the muscular walls of the heart. So long as the muscular fibres of the left auricle and left ventricle are healthy, the pulse remains regular, or the rhythm is only slightly altered.¹ The *sphygmographic characters* of the pulse may vary considerably in different cases of mitral regurgitation. The following tracings exhibit some of the alterations which are most frequently met with. (See figs. 186, 187, and 188).

2. *Accentuation of the pulmonary second sound.*—In free mitral regurgitation the pulmonary second sound is accentuated in consequence of the increased blood-pressure in the pulmonary artery. The degree of accentuation is to some extent indicative of the extent of the mitral lesion. It must, however, be remembered, that in attempting to gauge the extent of the mitral lesion, by the degree of accentuation of the pulmonary second sound which is present, due allowance must be made for the facts:—*firstly*, that increased blood pressure in the pulmonary artery may be due to any obstruction in the lungs, and therefore to pulmonary conditions which are

¹ Gaskell's observations as to the alterations in rhythm, which result from modifications in the condition of the cardiac muscle, quite independently of alterations in its nerve apparatus, are interesting and important as bearing upon this point. It must however, be remembered that, in man, there does not appear to be any direct connection between the muscular fibres of the auricle and the muscular fibres of the ventricle, at the auriculo-ventricular ring. (See figs. 182 and 183.)

associated with, but not caused by, the mitral lesion ; *secondly*, that the loudness of the pulmonary second sound, even in cases in which there is great obstruction to the passage of the blood through the lung, the result of advanced mitral disease or any other condition, depends, to a considerable extent, upon the condition of the right heart. When, for instance, the right ventricle is feeble, or the tricuspid valve incompetent, accentuation of the pulmonary second sound may not be observed.



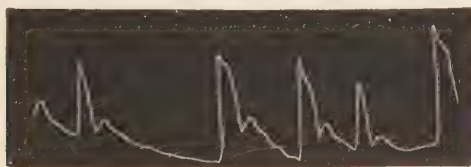
Pressure 3 oz.

FIG. 186.—*Mitral Regurgitation*.—M. A. C., æt. 16, admitted to Newcastle Infirmary 24th January 1878, suffering from cough and shortness of breath, dating from an attack of rheumatic fever two months previously. Heart's action very rapid (120-130). The first sound appeared to be reduplicated ; a systolic murmur was audible at the apex when the heart became slower.



Pressure 4 oz.

FIG 187.—*Mitral Regurgitation*.—S B, æt. 58, admitted to Newcastle Infirmary 25th February 1878, suffering from cardiac dropsy. There was a well-marked mitral systolic murmur, which disappeared under treatment. The heart was considerably enlarged (hypertrophied and dilated).



Pressure 3 oz.

FIG 188.—*Irregular and Intermittent Pulse*.—O. M., æt. 40, admitted to the Newcastle Infirmary suffering from cardiac dropsy. The heart was very much enlarged ; apex beat $4\frac{1}{2}$ inches below and 3 inches outside left nipple ; systolic mitral murmur.

3. *Physical signs indicative of alterations in the right*

heart.—Hypertrophy, dilatation of the right ventricle, tricuspid incompetence, and dilatation of the right auricle, are the secondary alterations in the right heart which may be produced by a mitral lesion. In *hypertrophy of the right ventricle*, the area of cardiac dulness, over the lower end of the sternum and adjacent costal cartilages, is increased; the impulse of the right heart is strong; the first sound of the right heart long, and the pulmonary second sound loud. In *dilatation of the right ventricle*, the area of cardiac dulness over the lower end of the sternum and adjacent interspaces, is also increased, but the impulse of the right heart is feeble; the first sound short and valvular, or replaced by a systolic murmur; when tricuspid incompetence is present, the second pulmonary sound may not be accentuated. In tricuspid incompetence, a systolic murmur is audible in the tricuspid area, and when the incompetence is great, true jugular, and (in some cases) hepatic pulsation is observed. *Dilatation of the right auricle*, which is usually associated with well marked evidence of tricuspid regurgitation, may give rise to increased dulness on percussion, and in some cases to increased cardiac impulse, in the neighbourhood of the third right interspace.

4. *Physical signs resulting from changes in the left ventricle*.—In free mitral regurgitation there is usually, as we have previously seen, some hypertrophy and dilatation of the left ventricle. In *hypertrophy*, the apex beat is displaced downwards and outwards; the area of cardiac dulness over the left heart is increased; and the impulse of the left heart is strong; the mitral murmur is loud and well propagated; the pulse of fair volume, tension and regularity. In *dilatation*, the apex beat is also displaced downwards and outwards, and the area of dulness over the left ventricle increased; but the cardiac impulse is weak, the pulse is small, feeble, and markedly irregular; the mitral murmur faint, or (in some cases) inaudible.

The characters of the cardiographic tracing in mitral regurgitation differ very considerably in different cases and vary with:—(1) The amount of the regurgitation; (2) the

condition of the left auricle (whether hypertrophied or merely dilated); and more particularly (3) the condition of the left ventricle, whether hypertrophy or dilatation predominates.¹

When the regurgitation is free, and more especially when it is uncombined with stenosis, the duration of the diastolic portion of the cardiac tracing is shortened. The eminence *k* is usually well marked; and the rapid flow of a large quantity of blood from the over-distended left auricle and pulmonary veins into the left ventricle may be manifested by a rapid ascent of that portion of the tracing placed between the eminence *k* and the rise which marks the commencement of the ventricular systole. In those cases in which the left auricle is hypertrophied rather than dilated, the eminence in the tracing which represents the auricular contraction is unusually well marked.

The character of the systolic portion of the tracing depends upon the condition of the left ventricle. When hypertrophy predominates over dilatation, the breadth of the systolic portion may be considerable. (See fig. 191.) When dilatation is in excess, the summit of the systolic part of the tracing is pointed, the sudden rise which corresponds to the first part of the ventricular systole being followed by an unusually rapid fall. (See fig. 190.) In some cases, the summit of the systolic portion of the tracing is double or forked. (See fig. 189.) Dr Sansom explains this forking of the apex, by supposing that 'after the first ascent of the lever, due to the hardening and rounding of the ventricle, there is a fall, because the ventricle has lost the point *d'appui* afforded by the stretched curtains of the normal valve; the continuing contraction of the ventricle, however, renews the elevation at the end of the systole.'² In some cases, the regularity of the systolic portion of the tracing is interrupted by a number of small indentations which represent the vibrations produced by the regurgitant blood current. (See fig. 189.) In many cases of this description a thrill can be felt when the hand is placed over the position of the murmur.

¹ The significance of the different portions of the cardiographic tracing is explained in the appendix.

² *Diagnosis of Diseases of the Heart*, p. 284.

The following tracings represent some of these points :—

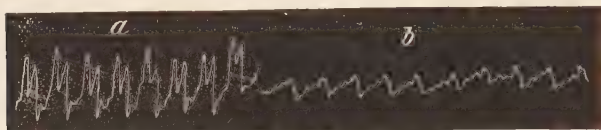


FIG. 189.—Cardiogram in a case of mitral regurgitation.—(After Sansom.)

'a, taken at the exact apex ; b, taken in the area of the loud systolic murmur. The diastolic portion of the tracing a is shortened ; the summit of the systolic portion is forked. A series of indentations representing the sonorous vibrations of the murmur are seen in b.'—(*Diagnosis of Diseases of the Heart*, p. 283.)

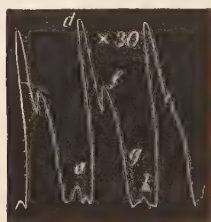


FIG. 190.

FIG. 190.—Cardiographic tracing from a case of mitral regurgitation combined with exophthalmic goitre.—(After Galabin.)

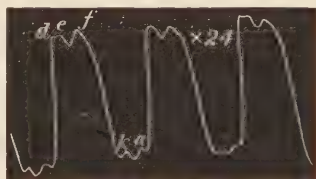


FIG. 191.

The duration of the diastolic portion of the tracing is shortened ; the eminence *k* is prominent ; the auricular impulse *a* present ; the systolic portion is indicative of dilatation of the left ventricle.—(*Guy's Hospital Reports*, 1875, p. 314.)

FIG. 191.—Cardiographic tracing from a case of mitral regurgitation.—(After Galabin.)

'A loud systolic murmur was present at the apex, preceded by a very faint rumbling sound. A presystolic murmur had been previously heard. The heart was much hypertrophied.'—(*Guy's Hospital Reports*, 1875, p. 313.)

5. The physical signs, which result from altered conditions of the peripheral organs, such as the lungs, liver, stomach, spleen, kidneys, etc., need not be detailed.

Diagnosis.—When a supposed case of mitral regurgitation comes under observation, the physician has to inquire—

Firstly, If the mitral valve is actually incompetent ?

Secondly, If the case is one of mitral regurgitation, is the incompetence due to muscular or relative incompetence, or to organic changes in the valve segments ?

Thirdly, If the regurgitation is due to muscular or relative incompetence, is the condition curable or not ?

Fourthly. If the regurgitation is caused by organic changes in the valve segments what is the extent and gravity of the lesion ?

Step No. 1. Is the mitral valve actually incompetent ?

There is seldom any difficulty in deciding this point ; for, in the great majority of cases of mitral regurgitation, a systolic murmur, having its point of differential maximum intensity in the mitral area, and its direction of propagation outwards towards the left axilla, is observed ; such a murmur is, I believe, quite characteristic of mitral regurgitation. Two sources of error must, however, be mentioned, viz.:—

(1) *The murmur may be wanting or inaudible.*—When the left ventricle is contracting feebly, the regurgitant blood current is not sufficiently powerful to produce an audible fluid vein or murmur. When, again, the heart is contracting very quickly, it may be difficult or impossible to detect the murmur. Now, feeble ventricular contraction and very rapid action of the heart are frequently observed in the later stages of mitral incompetence. There is, however, seldom any great difficulty in coming to a correct conclusion as to the nature of such a case. All the symptoms and signs of pulmonary and systemic venous engorgement, and of the secondary dilatation of the right and left hearts, which I have previously detailed, are present.¹ After a few doses of digitalis the frequency of the heart's action is lessened, the left ventricle regains power, and the murmur becomes audible.

(2) *The murmur may be simulated by other murmurs.*—Aortic, tricuspid, and pulmonary systolic murmurs can usually be distinguished from mitral systolic murmurs without difficulty. In doubtful cases attention must be particularly directed to:—the exact position of maximum intensity of the murmur, the exact direction in which it is propagated, the condition of the right and left ventricles, and the characters of the pulse.

¹ Evidence of a dilated condition of the left heart is of great diagnostic value in cases of this description, which are very closely simulated by primary lung affections with secondary alterations in the right heart and systemic venous circulation.

Pericardial murmurs audible at the apex can usually be distinguished by their rhythm, sound characters, area of distribution, and by the modifications which can be produced in them by the pressure of the stethoscope. Dr Sansom¹ states that in children it is not always possible to make the distinction. (See also table III., page 330,¹ in which the differential diagnosis is further set forth.)

Step No. 2.—The case is one of mitral regurgitation; is the incompetence muscular or relative, or is it due to organic changes in the valve segments?

In attempting to decide this point it must be remembered, that the two conditions (muscular and relative incompetence on the one hand, and incompetence due to organic changes in the valve segments on the other) are sometimes associated in the same case. An absolute distinction cannot, therefore, be made in every case. In many cases, the question can, however, be decided with considerable or absolute certainty; and this step in the diagnosis is one of the greatest practical importance, for, incompetence due to organic changes in the valve segments is a permanent condition, whereas regurgitation due to muscular and relative incompetence is in many cases completely curable. As a matter of practical experience, we know that this question presents itself for solution, more particularly in the following conditions:—

(1) *In acute cases attended with fever.*—In these cases, we have frequently to decide whether an apex systolic murmur is due to acute endocarditis or to acute relaxation, so to speak, of the cardiac muscle (acute muscular incompetence).

In acute rheumatism, in the course of which acute endocarditis is so apt to arise, it may be very difficult or impossible to come to a positive conclusion, as I have already pointed out in detail. (See p. 374.)

In other febrile conditions, such as typhoid and typhus, in the course of which endocarditis is rare, but acute febrile degeneration of the cardiac muscle common, the question is more easily decided. The period of the attack at which the

¹ *Lettsomian Lectures*, p. 38.

murmur is developed, and the presence or absence of symptoms indicative of mechanical interference with the course of the circulation, are the points to which attention must be particularly directed in making the diagnosis. When the murmur appears late in the disease (*i.e.* after a sufficient period has elapsed for the production of acute febrile degeneration of the cardiac muscle); when there are no distinct signs of mechanical derangement of the circulation, such as shortness of breath, dropsy, etc.; and when there are no embolic symptoms, a diagnosis of acute muscular incompetence may be confidently made.¹

(2) In *chorea* the same question, *i.e.* whether a mitral systolic murmur is due to acute endocarditis or to muscular incompetence, also occurs. But I need not further refer to this point, which has already been considered in detail. (See p. 773.)

(3) *In many chronic affections.*—In chronic cases, more specially *in those conditions which are associated with anæmia*, the question constantly arises whether a systolic mitral murmur is due to an organic lesion of the valve segments or to muscular incompetence.

In some cases of anæmia, the diagnosis is difficult or impossible. When, for instance, a patient who has had one or more attacks of rheumatic fever and who is distinctly anæmic, is found to be suffering from mitral regurgitation presenting the usual characters of regurgitation due to muscular incompetence, it is difficult or impossible to be quite certain that there are no organic changes in the valve segments. Again, in cases in which the patient is known to be the subject of chronic mitral regurgitation the result of organic changes in the valve segments, and is at the same time markedly anæmic, it may be difficult or impossible to decide what proportion of

¹ It is important to remember that in some cases of this description (typhoid, typhus, etc.), shortness of breath and cough may be due to lung complications, independently of any mitral lesion; and that some swelling of the feet may result from simple debility. It is therefore necessary, before attaching much importance to those symptoms, to be satisfied that the shortness of breath depends upon cardiac causes, and not upon (primary) lung disease; and that the dropsy is considerable and progressive.

the incompetence, so to speak, is due to the old organic lesion of the valve segments, and what proportion to the recent anæmic changes in the cardiac muscle. The majority of cases, which come under observation, are fortunately more simple, and in them the question can usually be determined by attention to the following points:—

1. *The general condition of the patient.*—In cases of muscular incompetence due to anæmic degeneration of the cardiac muscle, the general appearance of the patient at once attracts attention. In chlorosis and progressive pernicious anæmia, which are the affections in which this question of diagnosis chiefly arises, the extreme pallor of the mucous membranes, the lemon yellow colour of the skin, the fact that there is no emaciation so far as the subcutaneous fat is concerned, and the naked eye and microscopical characters of the blood, are very striking features, and show that a distinct cause of cardiac muscular degeneration is present.¹

2. *The condition of the heart and pulse, as determined by physical examination.*—A mitral murmur due to muscular incompetence, the result of anæmia, is usually preceded, and, I believe, almost always accompanied by a basic systolic murmur,—a pulmonary systolic murmur, or, more rarely, an aortic systolic murmur, or both. It is also accompanied by a venous hum in the neck.

Palpation and percussion show that the heart is dilated rather than hypertrophied.²

There is marked irritability of the cardiac muscle, the effect of which is seen in the sharp and easily excitable condition of the cardiac contractions, and by observing the characters of the pulse. This ‘celerity’ and irritability of cardiac action is noticeable during the whole course of the case though it is probably more marked in the earlier stages.

¹ As a matter of practical experience, we know that in cases of this description the cardiac muscle is always fatty.

² I am obliged to differ from those authorities who state that the heart is not dilated in progressive pernicious anæmia, for in all the fatal cases of that disease which have come under my own observation, there was more or less dilatation of all the cardiac cavities. The dilatation is, however, never so great, as we frequently see it, in fatal cases of organic disease of the valve segments.

In the earlier stages of anæmia the tension of the pulse is increased, as Dr Broadbent and others have pointed out. Dr Sansom¹ very justly, I think, states that in this fact we have an important differentiating mark between mitral regurgitation, the result of organic changes in the valve segments, and that due to anæmic degeneration of the cardiac muscle. It must, however, be remembered that in the later stages of progressive pernicious anæmia the increased tension of the pulse may completely disappear, as is well shown in the following tracing taken from a case of progressive anæmia, which died under my own care.

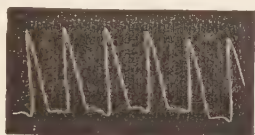


FIG. 192.—Pulse tracing in progressive Pernicious Anæmia.

R. R—, æt. 17, admitted to Newcastle-on-Tyne Infirmary, 21st February 1878, and died, notwithstanding the systematic administration of arsenic, on April 12th, 1878. This tracing was taken on March 19th; the artery is almost empty during diastole.

3. *The character of the murmur.*—The sound characters and extent of propagation of the murmur cannot be absolutely relied upon for diagnostic purposes. It may be stated, however, as a general rule, that a murmur which results from muscular incompetence is usually faint, and not well conducted; while a murmur due to organic changes in the valve segments, is, usually, in the earlier stages of the case² (*i.e.* while the left ventricle is powerful) loud and well propagated. Murmurs due to muscular incompetence are usually inaudible at the inferior angle of the left scapula.

¹ *Lettsomian Lectures*, p. 43.

² In consequence of the feeble action of the left ventricle, degeneration of its muscular fibre, etc., an organic murmur, which in the earlier stages of the case was loud and distinctly heard at the inferior angle of the left scapula, may become soft and inaudible in the back.

4. *The severity of the symptoms.*—In cases of anæmia, symptoms, due to venous engorgement of the peripheral organs and tissues, are much less prominent than in cases of organic disease of the valve segments in which there is the same amount of cardiac degeneration and dilatation.

5. *The effects of treatment.*—Chlorosis and other forms of simple anæmia can be speedily cured and the heart restored to its normal condition by the use of iron and other suitable remedies. In many cases of pernicious anæmia a perfect cure follows the systematic administration of arsenic. Whereas in mitral regurgitation due to organic lesions of the valve segments, although the symptoms may in most cases be relieved by appropriate treatment, the mitral regurgitation, and therefore the murmur, always remains; in fact, with the relief of the symptoms the murmur frequently becomes louder and better marked in consequence of the increased strength which the ventricular muscle acquires as the result of treatment.

6. *The history of the case.*—A history of acute rheumatism would, in a doubtful case, be in favour of organic diseases of the valve segments rather than of muscular incompetence. Too much weight must not, however, be given to this fact.

In cases of exophthalmic goitre the same difficulty in diagnosis also occurs. A systolic mitral murmur in this affection is generally due to the muscular or relative incompetence rather than organic disease of the valve segments. Each case must, however, be judged on its own merits, in accordance with the general principles just laid down.

In cases of aortic regurgitation it may also be difficult or impossible to say whether a mitral systolic murmur, which develops in the later stages of the disease, is due to muscular and relative incompetence, or to organic changes in the valve segments; in most cases it is, I believe, caused by the former condition (muscular or relative incompetence). This point is not one of much practical importance, for aortic regurgitation complicated with either form of

mitral incompetence is an extremely serious and incurable affection.

Step No. 3.—The regurgitation is due to muscular or relative incompetence ; is the condition curable or not ?

Step No. 4.—The regurgitation is due to organic changes in the valve segments ; what is the extent and gravity of the lesion ?

Both of these questions (Steps 4 and 5) are intimately connected with the prognosis, which I will therefore now consider.

Prognosis.—In considering the prognosis of mitral regurgitation, it is essential to draw a line of distinction between cases of muscular and relative incompetence on the one hand, and incompetence due to organic changes in the valve segments, on the other. The former condition is often completely curable ; the latter cannot be removed by any therapeutic measures with which we are at present acquainted.

The prognosis of mitral regurgitation produced by muscular and relative incompetence. Having decided that the case is one of muscular or relative incompetence, we must next endeavour to determine whether the condition is curable or not. The point is obviously one of the greatest practical importance, and in many cases it can be determined with considerable certainty. The whole question turns on the nature of the cause of the muscular debility ; the mitral regurgitation itself is altogether secondary in importance. In cases of this description, we know as a matter of practical experience, *firstly*, that if we can cure the muscular degeneration the mitral incompetence will disappear ; and *secondly*, that in many cases the muscular degeneration can be completely removed by treatment, while in others it cannot.

In a case of mitral regurgitation due to anæmic changes in the cardiac muscle, for example, we must endeavour to

determine what is the cause of the anæmia. If we come to the conclusion that the case is one of ordinary chlorosis, we can with great certainty predict that the condition will be speedily and completely cured. If, on the other hand, we decide that the case is one of progressive pernicious anæmia, we know that in some cases the termination will be fatal. In cases of progressive anæmia we can only, as I shall afterwards point out, judge of the curability of the case by watching the therapeutic results.¹

It is quite impossible to consider here in detail the prognosis of the many different primary conditions (such as the acute febrile affections, the different forms of anæmia, exophthalmic goitre, etc.), which may be attended with relaxation and degeneration of the cardiac muscle, and therefore with muscular and relative incompetence of the mitral orifice. The prognosis of some of these conditions, more particularly those in which the muscular degeneration is confined to the walls of the heart, will be again referred to when treating of the diseases of the myocardium. The points I wish to emphasise now are that, the mitral incompetence is in cases of this description of secondary importance, and that the object of the physician must be to ascertain the exact cause of the muscular debility or degeneration. It is only after this point is ascertained that a rational and scientific prognosis can be given.

The prognosis of mitral regurgitation due to organic changes in the valve segments.—Having come to the conclusion that the regurgitation is due to organic changes in the valve segments, we must next endeavour to determine what is the gravity of the lesion, and in trying to solve this question it is important to remember that the extent and the gravity of the lesion are by no means synonymous terms. It is only, when other things (such, for

¹ I say with *considerable* certainty, for it is, I believe, sometimes impossible to distinguish cases of chlorosis and of progressive pernicious anæmia, except by the therapeutic method. Cases of ordinary chlorosis are speedily cured by the preparations of iron (such as Blaud's pills), which are quite useless, and indeed often do harm in the 'idiopathic' form of the disease.

instance, as the general vitality and resisting powers of the patient) are equal, that the severity of the case varies directly with the amount of the regurgitation.

In judging of the gravity of the case, we have therefore to take into consideration the following points :—

1. The amount of the regurgitation.
2. The capabilities of compensation, and the powers of resistance possessed by the particular patient under observation.
3. Whether the lesion of the mitral valve is progressive or stationary.
4. The presence or absence of complications.

Let us consider each of these points in detail.

The amount of the regurgitation.—The mere characters of the murmur give us no information as to the amount of the regurgitation. It is important to direct attention particularly to this point, for it is sometimes supposed that a loud murmur, which is well propagated, is indicative of more serious disease than a soft, localised bruit. In many cases, the reverse is the fact ; indeed, we often find, as I have previously pointed out, that a murmur which, in the earlier stages of the case, was loud and well propagated, may, with the appearance of grave symptoms, become soft and faint, or disappear altogether ; the increased severity of symptoms, and the diminished loudness of the murmur, both being due to failure of the muscular power of the heart.

In order to form an opinion as to the amount of the regurgitation, we must take into account :—

Firstly, the extent of the secondary changes produced in the heart itself ; and *secondly*, the amount of mechanical derangement produced in the arterial and venous circulations respectively.

The degree of accentuation of the pulmonary second sound, the amount of the secondary hypertrophy and dilatation of the right heart, and the severity of the lung symptoms, are the points to which attention is to be directed in judging of the severity of the pulmonary congestion.

The condition of the systemic venous system, as determined by physical examination, the extent of the dropsy and of the other secondary effects of systemic venous engorgement, enable us to determine the extent of the obstruction to the venous return through the systemic veins.

The condition of the radial pulse, more particularly when compared with the size and strength of the left ventricle, gives information as to the extent of interference with the arterial circulation.

The patient's capability of resisting the disease.—The patient's power of bearing up under the lesion and resisting its bad effects depends upon :—

1. The power of compensation or resistance possessed by the heart itself.
2. The power of resistance possessed by the other tissues and organs.
3. His circumstances, habits, and surroundings.

1. *The capabilities of compensation or resistance possessed by the heart itself.*—We have previously seen, that compensation is for the most part effected by the production of secondary hypertrophy of the walls of the heart. (See p. 55.) Now, for the production of satisfactory hypertrophy, several conditions are necessary. In the *first* place, the muscular tissue of the heart must be healthy ; when it is diseased, when, for instance, it is in a condition of fatty or fibroid degeneration, the hypertrophy is never very satisfactory. In the *second* place, the muscle must receive a sufficient supply of healthy arterial blood. In the *third* place, the waste products of its combustion must be quickly removed ; and, in the *fourth* place, its trophic nerve apparatus must be healthy.

In endeavouring to ascertain *the condition of the cardiac muscle*, it is well to remember, that in chronic cases of mitral regurgitation, the lesion has generally been slowly and gradually progressing for months, it may be for years, before the patient comes under observation ; and that it is usually the failure of the compensation, which produces the symptoms and

which causes the patient to consult a physician. Now, in cases of this description, we are guided to a considerable extent in forming an opinion as to the condition of the cardiac muscle by the physical examination of the heart (*i.e.* by the size of the heart and of its different cavities, the character of its impulse), and by observing the condition of the venous and arterial systems. The following points must also be taken into consideration :—

(a) *The age of the patient.*—In youth the cardiac muscle is generally healthier, and the capabilities of repair are greater, than at more advanced periods of life.

(b) *The ætiology of the case.*—When the regurgitation has resulted from acute endocarditis, the capabilities of repair are generally greater than when it is due to atheroma or other degenerative changes. There are, however, many exceptions to this rule. An organic valvular lesion due to acute endocarditis is more frequently complicated, for example, with an adherent pericardium, the result of pericarditis, or with degeneration of the muscular wall of the heart, the result of endocarditis, than an organic valvular lesion due to other causes ; and these complications (adherent pericardium and fibroid degeneration, the result of myocarditis) add most materially to the gravity of the case.

(c) *The facility with which the cardiac muscle responds to the administration of cardiac tonics, such as digitalis.*—This is a point of great practical importance, for by administering cardiac tonics, and by watching their effects, we have an important means of ascertaining the condition of the cardiac muscle. In cases of fatty and fibroid degeneration, for example, the administration of digitalis often produces no beneficial effect.

In trying to ascertain *whether a sufficient amount of healthy blood is supplied to the cardiac muscle* we must observe :—

(a) The condition of the organs concerned in the manufacture and aeration of the blood, and in the separation from it of waste products, more particularly the condition of the stomach, liver, and kidneys.

(b) The effect of the lesion on the arterial system. If the

radial pulse is very weak and small, in consequence of the mitral disease, the strong probability is, that the circulation in the coronary arteries will also be extremely feeble.

(c) The condition of the coronary arteries. We have no direct means of ascertaining the condition of the coronary arteries. Atheroma of the superficial arteries, a dilated condition of the aortic arch, with or without disease of the aortic valves, an *arcus senilis*, and pains of an angina-like character are suggestive of atheroma of the coronary arteries; and when the coronary arteries are diseased, the supply of arterial blood to the cardiac muscle is, of course, still further interfered with.

The removal of the waste products of tissue change from the heart itself, is, of course, effected by the cardiac veins. When the return current through the cardiac veins is obstructed, the healthy nutrition of the cardiac walls must to some extent be interfered with. When therefore engorgement of the right heart and congestion of the systemic venous circulation, which are suggestive of impeded return through the cardiac veins, are present, the prospects of satisfactory hypertrophy are, for this and other reasons,¹ not very hopeful. (It must, of course, be remembered, that temporary engorgement of the right heart and venous circulation, the result of intercurrent pulmonary complications, such as acute bronchitis, is of frequent occurrence in the course of mitral disease.)

The condition of the trophic nerve apparatus connected with the heart probably has a very important influence in the production of satisfactory hypertrophy. The exact nature of this mechanism is not yet understood, though Dr Gaskell's researches seem to show, as I have previously pointed out (see page 33), that the vagus exerts some sort of trophic influence upon the cardiac muscle. Be that as it may, a healthy condition of the nervous system, and a serene placid and happy disposition exert a most important influence on the course of all cardiac cases, and undoubtedly aid in the production of satisfactory compensation.

¹ An engorged condition of the right heart and of the systemic venous circulation are indicative of an advanced mitral lesion.

2. *The power of resistance possessed by the peripheral tissues and organs.*—Here, as in the heart, the capability of resisting and bearing up against the effects of the lesion depend upon : (1) the condition of the tissue itself ; (2) the proper supply of nutrient material ; (3) the adequate removal of waste products ; and (4) the condition of its nerve supply.

The presence of complications, therefore, more especially of diseased conditions of the stomach and other organs which manufacture the nutrient fluid ; of the kidneys and other organs which purify it ; and of the nervous system which exerts such a powerful influence on all the processes of nutrition and resistance, are most important factors in determining the prognosis in cases of mitral regurgitation.

3. *The patient's circumstances, habits, and surroundings* also exercise an important influence on the progress of the case. —Patients who are obliged to follow laborious occupations, who are worried by financial or other matters, who are badly housed, badly fed, and badly clothed, who are unable to obtain medical advice, or who will not or cannot carry out the directions of their medical attendant, who are exposed to the vicissitudes of the weather, and who are given to excesses of any kind, succumb to a mitral lesion much more quickly than others who are more favourably situated. In them the lesion progresses more quickly, the capabilities of resistance are not so great, compensation more quickly fails, and complications on the part of the respiratory organs, for example, are much more apt to arise.

4. *The progressive or stationary character of the lesion.*

This is an extremely important point, and is determined by :—

(a) The history of the case.

(b) Close observation of the case and noting the condition of the patient from time to time.

(c) By comparing the duration of the case and the effects which the lesion has already produced on the heart and circulation—the progress of the symptoms, etc.

(d) The nature of the morbid process ; mitral regurgitation

caused by atheroma, for instance, will be more likely to progress rapidly than incompetence due to endocarditis. It is difficult, however, to lay down any general rule on this point.

The associated pathological conditions.—The important influence which complications exercise upon the course of mitral regurgitation has already been pointed out in speaking of the patient's power of resistance, and need not, therefore, be again referred to.

The advisability of communicating to or withholding from the patient the knowledge that the heart is diseased, is a question which always requires careful consideration. In most cases, it is, in my opinion, highly desirable to deal frankly, though of course the very reverse of abruptly, with the patient; for unless he realises the fact, that his heart is organically diseased, he cannot intelligently carry out our instructions as to treatment, and he does not guard himself so carefully, as he otherwise would do, against many things, such as over-exertion, exposure to cold, etc., which aggravate the disease or induce complications. In some cases, the communication should be made in a very guarded manner; and in the case of very nervous and easily depressed people, it is occasionally, though rarely, desirable to withhold the information altogether. In cases of this description, the physician should take care to protect himself against accidents, by communicating the exact condition of the patient to some judicious relative, for should this communication not be made, and the fact that the heart is affected be subsequently discovered (by some other physician, for example, or by the death of the patient), it may be supposed that the cardiac lesion was not recognised. In all cases of mitral regurgitation in which the patient is informed of his condition, he should be impressed with the fact that the lesion has little or no tendency to result in sudden death.

Treatment.—The *first indication* for the treatment of mitral regurgitation, and indeed of all diseases, is to effect a cure by removing the cause of the disease.

When the regurgitation is due to 'muscular' and 'relative' incompetence, the debilitated or degenerated condition of the cardiac muscle, on which the regurgitation depends, can often be completely cured; when the incompetence is due to organic changes in the valve segments, this happy result cannot be attained, for we know of no therapeutic measures by which a sclerosed valve can be restored to its previous healthy condition. It is necessary, therefore, to describe separately, the treatment appropriate to each of the two forms.

The treatment of mitral regurgitation due to muscular and relative incompetence.

The incompetence, which is due to febrile changes in the cardiac muscle, almost invariably disappears during the course of convalescence, and does not call for any special treatment. The main indication is to restore the tone of the general health by suitable food, fresh air and general tonics, amongst which iron, quinine, and strychnine are most useful. In the earlier stages of convalescence, when the cardiac degeneration is most marked, the patient should be cautioned against sudden effort, such as quickly rising from the recumbent to the standing position, and against everything which is likely to increase the cardiac weakness, or to induce syncope; prolonged immersion in a warm bath, for example, is to be avoided—I have known alarming indications of cardiac failure produced by a Turkish bath in a patient convalescent from rheumatic fever—tobacco smoking should be very sparingly, if at all, indulged in, and sexual intercourse altogether avoided. These points are, however, so obvious, that I need not go into details.

When the incompetence is due to fatty changes in the cardiac muscle, the pathological change on which the fatty condition of the cardiac muscle depends must be combated.

In *chlorosis* and all the ordinary forms of anæmia, iron or a combination of iron and arsenic, are the most useful drugs. The diet should be light and nutritious; the patient should have plenty of fresh air and outdoor exercise. The ventilation of the sitting and sleeping rooms must be particularly

attended to. Any exhausting discharge must, of course, at the outset, be arrested.¹

In cases of pernicious anæmia iron is not only useless, but in some cases seems actually injurious. The systematic administration of arsenic—beginning with two or three drops, and gradually increasing the dose—is the only drug treatment which, in my experience, is likely to be attended with satisfactory results. Many cures, of undoubted cases of this (previously) intractable disease, have been published, since I first recommended the use of this drug in the year 1877. It is not, however, invariably successful. I myself have met with cases in which it has failed, and other observers have recorded the same experience. It is most successful when given early in the disease, and it must be *perseveringly administered in gradually increasing doses*. I know of no means by which we can distinguish the cases of progressive anæmia which are curable by arsenic from the cases which resist this plan of treatment. We can only, in fact, form a judgment of the probable effects of the treatment by observing the results. In addition to the administration of arsenic, the dietetic and general management of the case must be carefully attended to; but space will not allow me to enter into details with regard to the treatment of this and other special forms of disease (such as leucocythæmia, Addison's disease, etc.), in which anæmia is a prominent symptom. I must repeat, that the mitral incompetence, is in these cases, a point of altogether secondary importance, and does not call for any special plan of treatment. All our therapeutic measures must be devoted to the primary condition on which the debilitated and degenerated condition of the cardiac muscle depends.

In cases of exophthalmic goitre, muscular incompetence at the mitral orifice sometimes also occurs. Iron combined with digitalis, and arsenic are the drugs which I have found most useful in this affection. It is seldom, however, that they effect

¹ In cases of chlorosis, iron is best administered in the form of Bland's pills, the tincture of the muriate, or the saccharine carbonate. Arsenic, in the form of Fowler's solution, may be given in combination with iron wine (*Vinum ferri*) or with the tincture of the muriate.

a cure. Dr Sansom¹ has found great improvement follow galvanisation of the cervical sympathetic. He uses twenty to forty Leclanché elements, one pole being placed behind the lower jaw in front of the sterno-mastoid, and the other either at a corresponding point of the opposite side, or over the vertebra prominens, or above the sternum at the inner edge of the sterno-mastoid muscle. In these cases, and indeed in all conditions in which the cardiac muscle is debilitated and degenerated, the application of a galvanic or Faradic current to the vagus is likely, I think, to prove useful. The experience which I have had in this method of treatment does not as yet allow me to speak positively on the subject. I base the treatment on the opinion of Gaskell, that the vagus exerts a trophic influence on the heart.²

When the incompetence is associated with, or depends upon, increased tension in the systemic arterial system, one of the most important indications is to reduce the general blood pressure. In cases of this description alcohol must be absolutely forbidden, butcher meat must be taken very sparingly, purgatives must be periodically administered (according to Dr Broadbent a calomel pill is the best purgative for this purpose) and alkalis, such as bicarbonate and nitrate of potash, prescribed; iodide of potassium and chloral are often useful.

The treatment of mitral regurgitation due to organic changes in the valve segments and tendinous cords.—In considering the treatment of mitral regurgitation due to organic changes in the valve segments or tendinous cords it is important to remember:—

(1) That the chronic and sclerotic lesions of the valve segments cannot be removed by any method of treatment with which we are at present acquainted; and that our treatment must, therefore, be directed to maintaining the balance of compensation and enabling nature to remedy and resist the defect.

(2) That in some cases the lesion is completely stationary,

¹ *Lettsomian Lectures*, p. 46.

² See p. 33.

and of such slight extent that although its presence is demonstrated by the persistence of a systolic apex murmur, it is not attended with any evident secondary alterations in the heart itself, nor with any mechanical derangement of the circulation. In cases of this description, in which there are no symptoms, and in which the pulmonary second sound may be only slightly, if at all, accentuated, no special treatment is required. The patient must be directed to attend to the condition of the general health, to avoid cardiac strain, and exposure to cold and other conditions likely to produce acute rheumatism and endocarditis. The management of these cases is very similar to that of cases of *progressive* mitral disease *before the failure* of compensation.

(3) That in some cases in which the lesion is more serious and is progressive, the compensation is for a time so perfect that there is no mechanical derangement of the circulation and therefore there are no symptoms; that in such cases the compensation may ultimately fail, and all the serious symptoms of venous engorgement, which have been previously detailed, result.

(4) That mitral regurgitation due to organic changes in the valve segments is often associated with incompetence due to defective muscular closure of the valvular orifice (*i.e.* muscular and relative incompetence), muscular incompetence being especially common after the failure of compensation, when the walls of the left ventricle become degenerated, and its cavity dilated. When there is any reason to suppose that the ventricular muscle is at fault, and that muscular or relative incompetence is present, the treatment which has been previously recommended for those conditions must be adopted. In all cases of mitral regurgitation, for example, in which the patient is anæmic—even when organic disease of the valve segments is known to be present—it is a good general rule of practice to treat the anæmia by the administration of iron, arsenic, and the means to which I have previously referred.

Bearing the general statements in view, we may now proceed to consider the treatment of progressive mitral disease,

due to organic changes in the valve segments, *before* and *after* the failure of compensation, respectively.

The treatment of progressive mitral disease before the failure of compensation.—So long as the compensation is perfect, the administration of drugs which act directly upon the heart itself is uncalled for. It must not, however, be supposed, that in cases of this description all treatment may be dispensed with ; on the contrary, though drug treatment is seldom required, the general management of the patient must be most particularly attended to. The great objects which must be kept in view in the treatment are :—

(1) To maintain the tissues as a whole, and the cardiac muscle in particular in the highest state of health, so as to enable nature to keep up those secondary changes which compensate the lesion and to resist the evil effects of mechanical derangement of the circulation.

(2) To avoid everything which is likely to hasten the progress of the valvular lesion.

In order to accomplish these objects, it is essential in the *first* place, to keep the circulation as tranquil as possible, and to avoid everything which is likely to throw any strain upon the heart. If the patient has been in the habit of following a laborious occupation he should be advised to give it up and to get some light, indoor employment ; all sudden efforts, such as lifting heavy weights, hurrying for trains, etc., should be strictly prohibited ; gentle outdoor exercise is, however, decidedly beneficial ; the amount of exercise which is advisable must be determined by the special circumstances of each individual case ; there is little difficulty in deciding this point, for in mitral cases any over-exertion on the part of the heart at once makes itself felt in the form of shortness of breath ; any exercise may be safely indulged in, which does not cause shortness of breath, and which does not produce excessive fatigue, *vice versa* every kind of exertion which does produce shortness of breath or excessive fatigue should be prohibited.

For the same reasons everything which produces mental worry and anxiety is to be avoided. The patient's surroundings should be as bright and cheerful as possible, and he

should not be allowed to take too serious a view of his own condition. With this end in view it is generally advisable in the case of sensible and intelligent patients, to explain clearly the exact nature of the affection, the objects of treatment, the accidents and complications which may arise, and the manner in which they are to be guarded against. The fact that the disease has little or no tendency to cause sudden death should be impressed upon the patient; many of the laity, when they are told that their hearts are not quite sound, jump to the conclusion that they are affected with a disease which may at any moment prove fatal, and to persons of a nervous and anxious disposition this idea is often a source of perpetual and terrible anxiety. It is of the utmost importance, therefore, to assure the subjects of mitral regurgitation that they need have no apprehension of sudden death. Excèses of all kinds, more especially over-indulgence in alcohol, in tobacco, or *in venere*, should be strictly forbidden.

In the *second* place, the condition of the digestive and excretory organs must be carefully attended to. The diet should be nutritious, but easily assimilated; milk, farinaceous foods, well-cooked and tender vegetables, a small quantity of butcher meat, fish, poultry, and game, may be allowed; pastry, rich made-dishes, and too much butcher meat, are to be avoided.

In those cases in which there is a tendency to excessive fat formation, the amount of saccharine, starchy and fatty articles of food should be restricted. It is difficult to lay down precise rules as to the amount of each article which may be allowed, for in each case the needs of the individual organism must be taken into account. It is important, however, to remember that most persons, if left to themselves, err on the side of excess, and that affectionate and well-meaning relatives are very apt, through mistaken kindness, to do harm by over-feeding patients.

Alcohol is not necessary in this stage of the disease; persons who have been accustomed to the use of wine or other alcoholic stimulants may be allowed a small quantity of alcohol, but the quantity should be *strictly moderate*, a larger

amount is not only in itself hurtful, but by producing a tolerance on the part of the system, it robs us, in the later stages of the disease, of one of our most effective therapeutic means of arousing the failing heart to greater activity.

Over-indulgence in tea and coffee must also be prohibited. Smokers may be allowed a small quantity of mild tobacco, but the amount, as in the case of alcohol, should be strictly moderate.

The condition of the excretory organs, the bowels, liver, kidneys, and skin, must be carefully attended to.

An abundance of fresh air is eminently desirable. As has been stated above, out-door exercise, which neither produces shortness of breath nor fatigue, is to be recommended. It is of no less (probably of still more) importance to have plenty of fresh air indoors, the proper ventilation of the sitting and sleeping apartments is, in the case of mitral disease (and indeed of all diseases), a point of the very first importance.

The patient should be recommended to go to bed early, and to take a large amount of sleep.

In the *third* place, complications of all kinds must be guarded against. It is particularly important to avoid exposure to cold and wet, for the congested condition of the respiratory organs, which is usually present in cases of mitral disease, even when the compensation is apparently perfect, predisposes the patient to attacks of bronchial catarrh and other pulmonary affections.

In many cases of mitral disease there is also a predisposition to rheumatism—a complication which is, of course, to be specially dreaded in the case of patients who are already the subjects of chronic valvular lesions.

The clothing must be warm, but not too heavy ; the under-clothing, in particular, must be sufficient ; flannel should be worn next the skin.

The treatment of progressive mitral disease after the failure of compensation.—The general plan of treatment suitable before the failure of compensation should, so far as is possible, be

continued, unless there is any reason to the contrary, and the following additional indications are to be carried out :—

(1.) *To strengthen, and, when necessary, to stimulate the failing action of the heart by the administration of cardiac tonics and stimulants.*

Some of the most important cardiac tonics are digitalis, convallaria majalis, caffeine, casca, strophanthus, arsenic, iron, and strychnine.

Digitalis is especially valuable in mitral regurgitation, and is, in fact, *the* remedy in progressive cases, such as we are at present considering. It seems to act as a true cardiac tonic, regulating the rhythm, diminishing the frequency of the heart-beats, and strengthening the contractions of the cardiac muscle; while at the same time it exerts a tonic effect upon the muscular coat of the minute arteries, and produces an increase of the arterial blood pressure. The tincture and the infusion of the fresh leaves are the most satisfactory preparations; and in prescribing this powerful remedy, we must endeavour, as Professor Sydney Ringer points out, 'to obtain the greatest therapeutic effect with the smallest possible dose;'¹ five to ten drops of the tincture, and two to four drachms of the infusion, three times daily are, as a rule, amply sufficient. When it is desirable to produce a more rapid effect (as, for instance, in those cases in which, when the patient first comes under observation, the dropsy is already considerable or great, the cyanosis marked, the respiration much embarrassed, the urine scanty and loaded with lithates, the pulse very small, weak, and irregular, and the action of the heart tumultuous), much larger doses may be advantageously given.

During a course of digitalis treatment, the effects of the drug on the pulse, the urine, and the dropsy, must be carefully watched. So long as the urine remains scanty and deposits urates, full doses may be safely continued. After free diuresis is established, and more especially if the pulse falls below 70 the dose must be immediately reduced, or the administration of the drug suspended altogether. In those cases in which digitalis produces sickness, as it frequently does when given in

¹ *Handbook of Therapeutics*, p. 486.

too large quantities, or when too long continued, repeated small doses of brandy, or, still better, in my experience, of iced champagne, or a mixture of iced champagne and brandy should be prescribed.

Convallaria majalis.—This remedy has of late been strongly recommended, and seems to possess many of the beneficial properties of digitalis: it increases the force of the cardiac contractions, while it lessens the frequency of the beats; at the same time it produces free diuresis. It seems to be well borne; it exerts no prejudicial effect upon the digestive organs, but, on the contrary, rather increases the appetite and the action of the bowels. It may be given in the form of extract (5–8 grs. of Savory and Moore's extract three times daily), fluid extract (5–20 mms.), or tincture (5–20 drops).

Caffeine seems also to be a cardiac tonic, though not nearly such a powerful one as digitalis. It slows the action of the heart, while at the same time it increases the force of the cardiac contractions; its most powerful and useful effect in cases of mitral regurgitation is, however, its diuretic action, a point to which I shall frequently refer more in detail. Citrate of caffeine is a convenient preparation, and may be administered in 3–6 grain doses three times a day.

Casca.—Of this drug, which has been strongly recommended by Dr Brunton, I have had no personal experience.

Strophanthus.—This remedy, which has lately been extensively used by Professor T. R. Fraser, is a powerful cardiac tonic, similar in action to digitalis, but more energetic.¹

Arsenic is a most important cardiac tonic, and should be much more frequently and systematically prescribed in cases of mitral regurgitation than it is at present. It is especially useful in those cases in which the failure of compensation is accompanied by fatty changes in the cardiac muscle or pain in the region of the heart; it should be given in the manner already recommended in speaking of the treatment of muscular and relative incompetence. (See page 463.)

Iron, strychnine, and quinine, are all most useful in some

¹ Professor Fraser intends, I believe, to publish before long the results of his investigations on this drug.

cases of mitral regurgitation, though they can hardly be called true cardiac tonics in the same sense as digitalis.

Stimulants.—After the failure of compensation, alcoholic stimulants, ether, spirits of chloroform, carbonate of ammonia, etc., are often most useful, and require to be freely given. The application of fly blisters to the præcordial region is, also, in some cases an effective means of stimulating the flagging heart.

It must not be forgotten that the temporary administration of cardiac tonics and stimulants is often most useful and necessary in the earlier stages of the affection, *i.e.* before the failure of compensation; it is, in fact, impossible to draw any hard and fast line between the periods *before* and *after* compensation; the remedies which are useful in the one stage are consequently often required in the other; even when the compensation appears to be perfect, the reserve force possessed by the heart is very slight, and any temporary condition, such, for example, as an intercurrent attack of bronchial catarrh, which throws an increased strain upon the heart, may for the time upset the balance and necessitate the temporary use of cardiac tonics and stimulants.

(2.) *To relieve venous congestion and treat the pathological conditions and symptoms which result therefrom.*

This is a most important indication in the later stages of mitral disease. Space will not permit me to detail the special means which are required for each individual complication. I must content myself with referring to some of the more important. And in treating the venous engorgement and its results, we must never forget that the mechanical derangement of the circulation on which they depend is due to defective action of the cardiac pump, and that one most important means of treatment consists in the administration of cardiac tonics and stimulants. We may, then, lay it down as a general rule, that in treating the bronchial catarrh, dropsy, dyspepsia, and other conditions which result from venous congestion, digitalis or other cardiac tonics should, unless there is good reason to the contrary, be combined with the

drugs which are required for the special complication under consideration.

In addition to the administration of cardiac tonics and stimulants, we must endeavour :—*firstly*, to remove the venous engorgement by local and general measures ; *secondly*, to improve the nutritive condition of the blood, by (a) careful attention to the condition of the chylopoietic viscera ; (b) by promoting free action of the stomach, liver, intestines, kidneys, and skin ; and (c) by establishing a healthier condition (tone) of the nerve centres.

I shall now briefly refer to the special treatment of some of the more important symptoms which occur in the latter stages of mitral disease ; and for the sake of convenience, it will be well perhaps to commence at the lungs, and to proceed backwards in the course of the regurgitant blood current, rather than to take the symptoms in the order in which they are likely to arise, in the living patient.

Lung complications and symptoms.—The *dyspnœa* which is due to exertion does not of course require any other treatment than rest ; the continuous *dyspnœa* and *orthopnœa*, which are often so distressing in the later stages of mitral disease, are best dealt with by the administration of cardiac tonics and stimulants, and of remedies suitable for the lung complications (bronchitis, œdema of the lungs, hydrothorax, etc.) which happen to be present. If there is much venous engorgement, the application of dry cups over the back, of leeches over the præcordia, or venesection, are in some cases beneficial. The inhalation of oxygen and compressed air is strongly recommended by some writers, and seems to be useful, by counteracting the highly venous condition of the blood.

Cases of continuous *dyspnœa* and *orthopnœa*, in which there is often very great general exhaustion and insomnia, are often most materially benefited by the administration of morphia ; this drug, which is best given by subcutaneous injection, has been highly recommended by Clifford Allbutt, Sansom, and others, and has sometimes proved most useful in my own hands.

In the exceptional cases in which a mitral lesion and

severe dyspnœa are associated with high arterial tension, and in some cases in which Cheyne-Stokes' respiration occurs, nitrite of amyl should be inhaled during the attack itself; nitro-glycerine in small doses ($\frac{1}{200}$ th of a grain) may be given during the intervals; bromide of potassium and chloral I have found most useful in some cases of this description.

Bronchitis, œdema of the lungs, hydrothorax, pleurisy, pneumonia, and other lung complications.—Space does not permit me to enter into details with regard to the treatment of the complications. In all cases it is advisable to combine cardiac tonics, more especially digitalis and cardiac stimulants, with those remedies which the special complication in each individual case demands.

Hæmoptysis.—The bleeding is seldom so profuse as to require any special treatment; in some cases it seems rather to give relief than to be prejudicial; when the pulmonary apoplexy is followed, as it often is, with localised pleurisy or pneumonia, the local and general measures suitable for those complications must of course be employed.

Engorgement of the right heart.—When cardiac tonics and stimulants, purgatives and diuretics, fail to relieve the engorgement of the right heart, the application of a mustard poultice or blister to the præcordial region is, in some cases, beneficial. The most satisfactory means of relieving great engorgement of the right heart is, however, the abstraction of blood by leeches applied to the præcordia, or even by general venesection.

Engorgement of the systemic venous circulation.—In addition to the free use of cardiac tonics and stimulants, the administration of diuretics, diaphoretics, and especially purgatives which produce free watery evacuations, are the remedies on which reliance must be chiefly placed. Digitalis and caffeine are the most useful diuretics, and are advantageously administered in combination, as recommended by Brakenridge;¹ the acetate,

¹ Brakenridge thinks that caffeine has a direct action upon the secreting structures of the kidney, and does not produce its diuretic action as digitalis is supposed to do, entirely by increasing the blood pressure.—(*Edinburgh Medical Journal*, August 1881, p. 100, *et seq.*)

nitrate, and bitartrate of potash and squills are also serviceable. I have found nitrite of amyl a most valuable diuretic in some cases, more especially where the arterial tension has been high, and it has been desirable to produce rapid distention of the vessels of a congested kidney; the application of a warm poultice or of dry cups over the region of the kidney is often useful in cases of this description. Jalap, elaterium, and scammony are the most useful purgatives; purgatives which are apt to cause hæmorrhoidal irritation, are to be avoided.

The skin should be kept active by washing, rubbing, and bathing; in the later stages of the disease it is not, as a rule, advisable to immerse the whole body in the bath, but individual portions should be washed separately. It is important to remember that prolonged immersion in a warm bath often produces considerable depression; patients with mitral disease should therefore bathe judiciously.

Dropsy.—Subcutaneous dropsy, which is one of the earliest indications of progressive mitral disease, is to be treated:—

Firstly, By the internal administration of cardiac tonics and stimulants, diuretics and purgatives.

Secondly, By local measures. The swollen parts should, if possible, be placed in such a position that the return current of blood to the heart is facilitated; this is easily of course accomplished so long as the dropsy is limited to the lower extremities. When the dropsy becomes great, it may be necessary to evacuate the fluid either by simple puncture or by means of Southey's trocars; whichever method be adopted it is of the greatest importance to remember that erythema and low forms of inflammation are very apt to arise at the seat of the punctures, and that this is more particularly the case when the fluids which escape are allowed to remain in contact with the skin and to decompose; the greatest attention should be given to cleanliness, all sources of external irritation avoided, and decomposition, so far as is possible by antiseptic means, prevented.

Erysipelas and gangrene of the skin which sometimes

arise, are very serious complications. The local treatment must of course be conducted on general surgical principles; cardiac tonics and stimulants being at the same time freely administered.

(3.) *To improve the quality of the blood and attend to the condition of the chylopoietic and excretory organs.* It is particularly important to attend to the condition of the stomach; it must be remembered that this organ shares in the general venous engorgement, and that in the later stages of mitral disease, at all events, gastric catarrh and its resulting dyspepsia are almost invariably present;¹ the digestive powers of the stomach are consequently seriously impaired; the diet must, therefore, be regulated accordingly. When dyspeptic symptoms are prominent, small quantities of easily digestible food, such as milk, farinaceous foods, soups, raw eggs beaten up with a little brandy, should be given at frequent intervals; the mineral acids, strychnine, arsenic, and infusion of calumba are useful additions to the other remedies which have been previously recommended. It is sometimes advisable to supplement feeding by the stomach by rectal alimentation; enemata of defibrinated ox-blood or the prepared peptone enemata—consisting of beef, milk, and farinaceous food—recommended by Dr Sansom, are the most suitable and convenient forms; ‘from two to four ounces are injected slowly into the rectum, and repeated every three or four hours.’² I cannot too strongly insist upon the importance of careful regulation of the diet in all stages of progressive mitral disease.

The action of the liver must be encouraged by suitable purgative and cholagogue remedies.

The importance of procuring free intestinal evacuation, and of attending to the function of the kidneys, has already been insisted upon.

¹ Gastric catarrh and dyspepsia are often also present in the early stages.

² Dr Sansom’s *Lettsomian Lectures*, p. 44. (The author is speaking of the treatment of cases of progressive pernicious anæmia, but the same means are useful in some cases of organic mitral disease in which the functions of the stomach are seriously impaired.)

MITRAL STENOSIS.

Definition.—Narrowing of the mitral orifice.

Ætiology.—Stenosis of the mitral orifice is almost invariably due to permanent organic changes in the segments and base of the mitral valve; with rare exceptions these changes are very slowly and gradually developed.¹ It is essentially a disease of early life, and is, in my experience, most frequently developed (it would perhaps be more correct to say most frequently detected) between the ages of fifteen and twenty-five. Occasionally, though very exceptionally, it is congenital, and is then usually associated with congenital tricuspid stenosis. In a large proportion of cases it results from rheumatic endocarditis, but it would appear from Dr Sansom's observations² that, unlike mitral regurgitation, it is more frequently developed after mild rheumatic manifestations and in cases, in which articular phenomena were not manifested at all, than after severe attacks of rheumatic fever. Dr Sansom's observations also seem to show, that repeated attacks of acute rheumatism do not generally tend to produce the lesion. Mitral stenosis is much more frequent in females than in males, a circumstance which is probably to be explained by the facts that the endocarditis of early life is more likely to result in mitral stenosis than the endocarditis of the fully-formed adult, and that acute rheumatism is three times more prevalent in girls, between the ages of eleven and fifteen, than it is in boys at the same age. It is interesting to note that chorea is also much more frequent in girls than in boys. There is probably, as Dr Barlow has suggested, a distinct relationship between the two conditions.³

Morbid Anatomy and Pathological Physiology.—Mitral

¹ Occasionally narrowing of the mitral orifice is due to a mass of vegetations of recent formation; and in very exceptional cases it has been produced by the pressure of a tumour, the valve segments themselves being healthy.

² *Lettsonian Lectures*, p. 80.

³ Two theories may be advanced in order to explain the greater liability of the female sex to chorea and mitral stenosis, viz.:—(1) That the chorea and the mitral stenosis are both the result of rheumatism, which is, as we have seen, three times more common in girls between the ages of eleven and fifteen than in boys at the same age; and (2) that chorea, even when non-rheumatic, is frequently accompanied by endocarditis, which is the cause of the mitral narrowing.

stenosis is, as I have mentioned, almost invariably due to permanent organic changes. The exact appearances vary somewhat in different cases. In some, the constriction is chiefly due to the fact, that the orifice itself is narrowed by sclerotic and cicatricial changes in the fibrous ring which surrounds it. In cases of this description, the orifice, when seen from above, looks like a narrow slit,¹ hence the term button-hole mitral which has been applied to it. (See fig. 193.) The segments of the valve and chordæ are at the same time more or less rigid and thickened. In other cases the segments, chordæ and tips of the papillary muscles are all welded together into a dense mass of cartilagenous-like tissue, and project into the cavity of the left ventricle in the form of a hollow cone. A perfect cone of this description, such as is represented in fig. 195 is rare, but in the majority of cases of mitral constriction due to endocarditis, a tendency to this formation is observed. These two conditions, narrowing of the orifice and fusion of the segments, are generally associated.

In rare cases, the mitral segments are quite healthy, and the stenosis is due to the presence of large calcareous nodules in the muscular wall of the ventricle. (See figs. 196, 197.) Deposits of this description are usually associated with atheromatous degeneration of the arteries; in some cases they are gouty and consist of urates, in others they represent, I think, syphilitic gummata which have become calcified. In three cases of this description, which have come under my own observation, and in which the valvular orifice was very notably narrowed, there were no symptoms or signs indicative of the condition during life. Two of the patients were old people who lived tranquil lives; the third was a man who was for some time under my care, and who died from a large aneurism of the descending thoracic aorta. (See figs. 268 and 269.)

On microscopical examination, the thickened valve segments and chordæ tendineæ are found to present the histological characters of chronic endocarditis, which have been previously described.

¹ The healthy mitral valve, when seen from above, presents a slit-like appearance; in stenosis the slit is narrowed.



FIG. 193.—*Stenosis of the mitral orifice, seen from above. (Natural size.)*

The left auricle, which is considerably dilated, has been cut open; an ante-mortem clot (*b*) fills the appendix and projects into the cavity of the auricle. *a*, points to the stenosed mitral orifice; *c*, to the closed foramen ovale; *d*, to the outer and posterior surface of the left ventricle.

Note—In fig. 195 the appearance of the valve as seen from the ventricular side is shown.



FIG. 194.

Interior of the Left Ventricle showing (diaphragmatic) contraction of the Mitral Valve, and disease of the Aortic Cusps. Somewhat larger than the actual preparation (the ventricle, at the point of origin of the aortic cusps, measures in the drawing $3\frac{1}{2}$ in., and in the actual preparation $2\frac{1}{2}$ in.).

a, is situated on the anterior segment of the mitral valve; the mitral cusps, chordæ tendineæ and papillary muscles are welded together.

b, points to one of the diseased aortic cusps, it is bulged out towards the ventricle, its ventricular surface is roughened.

c, points to a small, rough depression (ulcer) on the exterior of the aortic cusp, b.



FIG. 195. *Stenosis of the Mitral Valve. (Natural size.)*

The segments of the mitral valve, the chordæ tendineæ, and the papillary muscles are, as it were, fused together into a dense, firm mass. The mitral orifice is extremely contracted, and will not admit the point of the little finger. The letter *a*, points to the base of the aorta and aortic valve flaps; *b*, anterior segment of mitral valve; *c, c*, thickened chordæ tendineæ and papillary muscles.

Note.—The auricular surface of the stenosed valve is represented in fig. 193.

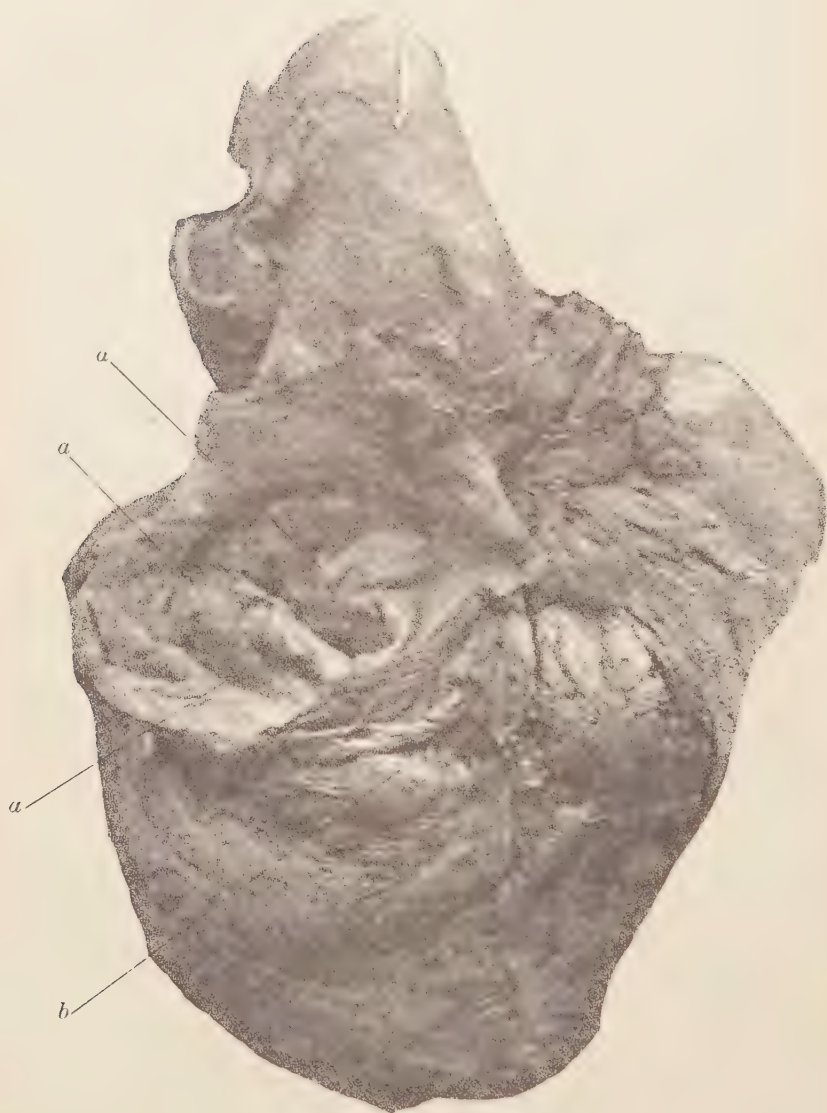


FIG. 196.—Heart in a case of mitral stenosis, in which the obstruction was due to the deposit of large calcareous masses in the base of the valve and adjacent walls of the heart. (Natural size, seen from behind.)

a, a, a, calcareous masses surrounding the mitral orifice, which is seen from above ;
b, calcareous mass in the wall of the left ventricle.



FIG. 197.—Section through the posterior walls of the left auricle and ventricle, and through the posterior segment of the mitral valve.

Stenosis of the orifice was produced by calcareous nodules, one of which (*a*) is shown in the figure, in the base of the valve; the mitral cusps were healthy; *b*, transversely divided wall of the left ventricle; *c*, interior of left auricle; *d*, papillary muscle, partly seen in section; *e*, posterior segment of mitral valve.

The loss of elasticity in the valve segments, generally renders the valve incompetent. Mitral stenosis then, in the earlier stages at all events, is usually accompanied by mitral regurgitation.

Pathological Physiology.—The *first* effect of mitral stenosis is to interfere with the passage of the blood from the cavity of the left auricle to that of the left ventricle. The left auricle empties itself with difficulty, its systole is prolonged, and blood accumulates and stagnates behind the obstruction; the left ventricle receives a smaller supply of blood than in health, and the arterial system is, in consequence, imperfectly distended. Stenosis, in short, produces more or less engorgement

behind, and more or less anæmia in front of the mitral orifice. The *second* effect is to produce a series of changes in the heart itself, the other parts of the circulation, and the peripheral organs, which, with the differences which I shall presently mention, resemble the secondary effects produced by mitral regurgitation. (See p. 428.)

The extent of the primary and secondary effects of mitral stenosis varies with :—the extent of the lesion, the degree of compensation, and the resisting power of the patient, and of his individual tissues. But since, in treating of mitral regurgitation, I have already considered in great detail the nature of these changes and the general principles which determine their production, I need only now refer to the points of difference between mitral stenosis and mitral regurgitation.

In the *first* place, then, mitral stenosis differs from mitral regurgitation in the fact, that the vascular engorgement of the left auricle, and consequently of the pulmonary circulation, is more continuous. In mitral regurgitation, the engorgement of the left auricle and pulmonary circulation is suddenly relieved by the occurrence of the ventricular diastole; and the blood which has been pent up, so to speak, in the cavity of the left auricle and in the lungs, during the ventricular systole, has no difficulty in passing onwards into the cavity of the left ventricle. But in mitral stenosis the obstruction persists, during the ventricular diastole. When the orifice is much stenosed (more especially in the terminal periods of the case, *i.e.* after the auricle has become dilated and its muscular wall paralysed¹), the left auricle is never emptied; clots are extremely apt to form in the appendix (see figs. 193 and 198), and even in the cavity of the auricle itself;² embolic

¹ Cases of extreme stenosis are occasionally met with, more especially in young subjects, in which the hypertrophy of the auricular wall continues good even until the end, and in which the cavity of the left auricle is very little dilated.

² It occasionally, though very rarely, happens, that the whole auricle becomes filled up with a laminated clot, a narrow channel only remaining for the passage of the blood through it. Only one case of this description has come under my own observation; in it the aortic orifice was also diseased (incompetent), and there was, therefore, in addition to this mitral stenosis, a further cause for stagnation and clotting in the left auricle.



FIG. 198.—*Mitral Stenosis* seen from above. (Natural size.)

The cavity of the left auricle has been cut open ; a mass of calcareous nodules (*a*), to which some recent vegetations are attached, surround the constricted orifice ; the appendix contains a thrombus (*b*).

symptoms, are therefore, more apt to arise in cases of mitral stenosis than in cases of mitral regurgitation. The dilated and permanently distended auricle sometimes exerts continuous and injurious pressure upon the surrounding parts; the left bronchus may for a time be compressed, and collapse of the left lung may result from this cause.

In the *second* place, mitral stenosis is essentially a disease of early life. The compensatory changes are, therefore, in many cases much more perfect than in the case of mitral regurgitation, which so frequently occurs in old people, and which is so often due to degenerative changes in the cardiac muscle.

In the case of mitral stenosis, compensation is partly due to hypertrophy and dilatation of the left auricle, but chiefly to secondary hypertrophy of the right ventricle. In young healthy persons the right ventricle is, for a long time, fully equal to the strain which is put upon it, and is able to propel the blood through the lungs, through the left auricle and through the stenosed mitral orifice with sufficient force to compensate the lesion; compensation being of course assisted by the hypertrophy of the left auricle, which also occurs. In mitral stenosis the systemic venous circulation may for a considerable time be protected by the hypertrophy of the right heart. The pulmonary circulation, on the contrary, is subjected to a severe strain, even in the earlier stages of the lesion; this arises from the facts, (1) that the obstruction in front is constantly present, *i.e.* both during the diastole as well as during the systole of the left ventricle, and that the suction action of the left ventricle, which facilitates, as we have already seen, the flow of blood through the lungs, cannot be so powerfully exerted as in health; and (2) that the hypertrophied right ventricle is pumping the blood from behind with increased force into the pulmonary circuit. In the earlier stages of mitral stenosis, then, the engorgement of the lungs is greater and more continuous than in the earlier stages of mitral regurgitation, a fact which explains the more frequent occurrence of hæmoptysis in stenotic cases. So long as the hypertrophy of the right ventricle is capable of forcing

the blood through the lungs, and so long as the systemic venous system is not much engorged, there is little derangement of the general health, the great functions of digestion and assimilation, excretion and innervation, being little, if at all, interfered with. Pulmonary symptoms and complications may, on the contrary, be prominent.

In the *third* place, the cardiac contractions, and therefore the pulse, are not so frequent in cases of mitral stenosis as in cases of mitral regurgitation. In the latter lesion, a large volume of blood is being constantly sent backwards and forwards between the cavities of the left auricle and left ventricle (the ventricle is, as it were, playing at battledoor and shuttlecock with the auricle, instead of forwarding all its contents into the aorta), both cavities are more quickly distended, and, therefore, contract more frequently than in health. In the case of mitral stenosis, on the contrary, the left auricle is, in consequence of permanent over-distention, being more constantly stimulated, and, so long as its muscular wall remains healthy, its contractions are more forcible and more prolonged than in health; the left ventricle, on the contrary, receives less blood than in the normal condition, it therefore requires less time to empty itself. As a *nett* result, the frequency of the radial pulse is, in the earlier stages of the case at all events, not increased as it is in cases of mitral regurgitation, indeed it is sometimes slower than normal; the tension of the pulse is, too, in the earlier stages, much less seriously impaired.

In the *fourth* place, the left ventricle does not become hypertrophied, as it does in mitral regurgitation. In fact, in many cases of mitral stenosis the left ventricle is somewhat atrophied, the diminished size of its cavity and thinning of its wall being due to the facts, that it receives less blood from the auricle, and has less work to do in forcing that blood into the aorta, than under normal circumstances.

These remarks, of course, apply to cases of mitral stenosis, in which there is little or no mitral regurgitation. It must, however, be remembered, that in the majority of cases of mitral stenosis some regurgitation is present, and that the amount of this regurgitation is sometimes considerable. In cases of this

description, the points of distinction between mitral stenosis and mitral regurgitation, which I have just detailed, may not be observed. The association of mitral regurgitation with stenosis probably explains the fact, that in some cases of mitral stenosis the left ventricle is hypertrophied rather than atrophied. In the later stages, hypertrophy of the left ventricle may possibly, as Friedreich has supposed, be due to the difficulty which the blood meets with, in passing from the arterial into the distended and engorged venous system; or, to venous engorgement of the wall of the ventricle, a condition which induces connective tissue overgrowth and some degree of apparent (false) hypertrophy. Again, in cases of mitral stenosis the left ventricle may be hypertrophied from causes outside the heart, such, for example, as cirrhosis of the kidney.

Clinical History.—The onset of mitral stenosis is, as a rule, very insidious. In some cases the condition is gradually developed after an acute attack of rheumatic fever; in others, and these possibly constitute the majority, there are no definite rheumatic symptoms or other signs of acute illness to mark the commencement of the disease.

Symptoms.—The compensation usually remains perfect for a considerable time, it may be for years; and it is only when the patient makes any extra exertion, that he feels short of breath, suffers from palpitation, and begins to suspect that there is something wrong with his heart. Pulmonary complications, such as bronchial catarrh and hæmoptysis are of frequent occurrence even during the stage of good compensation, and result, as I have already mentioned, from the engorgement of the lungs which is usually present even in the earlier stages of the case. After the failure of compensation, the systemic venous circulation becomes seriously embarrassed; dropsy and the other symptoms, which I have already described under the head of mitral regurgitation (see p. 440) are then developed. Accidental symptoms, due to embolic plugging of some distant arterial trunk, are not uncommon; the

embolon may be carried to almost any part of the body, but the vessels which are most frequently plugged are the renal, splenic, and left middle cerebral arteries.

The exact nature of these accidental symptoms depends, of course, upon the vessel which happens to be obstructed, and to some extent upon the size of the plug. *Obstruction of the left middle cerebral artery* causes right-sided hemiplegia (paralysis of the face, arm, and leg, on the right side) and aphasia. *Obstruction of the splenic artery* is usually attended with some pain in the region of the spleen, which, on physical examination, is found to be enlarged and tender to the touch. *Obstruction of the renal artery* may be attended with pain in the back, with albuminuria or hæmaturia. *Obstruction of a large branch supplying the intestine* is generally accompanied by severe spasmodic colic, and is sometimes followed by diarrhœa. In each of these cases there is usually, as Dr Sansom¹ and others have pointed out, some elevation of temperature; in fact, the sudden onset of pyrexia in a patient who is affected with mitral stenosis, is, in the absence of any obvious cause, strongly suggestive of an embolic infarction.

Physical Signs.—A presystolic murmur having its point of differential maximum intensity in the mitral area is, according to most authorities, pathognomonic of mitral stenosis,² and is, therefore, the most important sign of the disease.

The murmur is usually rough and rolling, grinding or churning in character. It is almost exactly simulated, as Balfour points out, by the sounds which are produced when 'the symbols Rrrb or Vööt are vocalised.' It occurs towards the end of the long pause, and ceases abruptly with the occurrence of the first sound. It is usually very localised, and is best heard at the left apex of the heart or at a point slightly

¹ *Lettsomian Lectures*, p. 93.

² Professor Austin Flint differs, as I have previously mentioned, from this—the usually accepted—view; and thinks that a presystolic murmur does not necessarily indicate an organic lesion of the mitral valve. Dr Sansom also states, that in some cases of aortic regurgitation a presystolic murmur is heard at the apex of the heart, but he does not, so far as I am aware, endorse the opinion of Professor Austin Flint, that it is generated at the mitral orifice.

within the left apex; it is propagated directly towards the apex, and, therefore, directly, as it were, into the stethoscope and ear of the observer. (See fig. 199.)

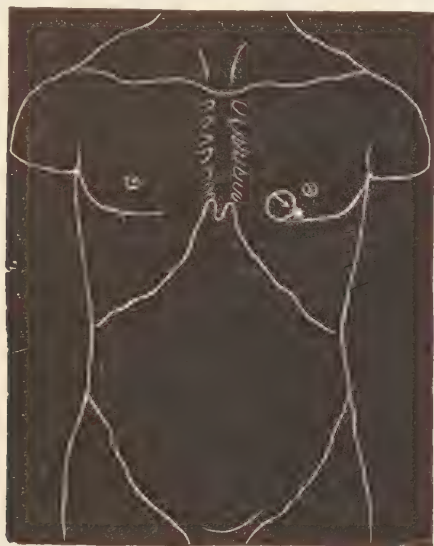


FIG. 199.—Outline figure showing point of differential maximum intensity (*) of the presystolic mitral murmur (mitral stenosis); and the direction in which it is propagated. (The murmur is often best heard a little above and internal to the apex-beat, which in the diagram corresponds to the star. *)

It is important to remember that this murmur is not present in all cases of mitral narrowing. In order that it may be produced, the conditions necessary for the formation of an audible fluid vein must be present; in other words, the orifice must be sufficiently constricted, and the blood current passing from the left auricle to the left ventricle must be driven through the constricted orifice with sufficient force.

Now, in the earlier stages of mitral stenosis (*i.e.* in cases, for example, in which a slight degree of stenosis is found after death), a presystolic murmur is often absent owing to the fact that the orifice is not sufficiently constricted to produce a fluid vein. In cases of this description the valve is usually incompetent, and, during life, the case is characterised by the usual symptoms and physical signs of mitral regurgitation.

Again, in the later stages of the disease, when the left auricle is much dilated, when its muscular wall is weak and degenerated, or when, as in some rare cases, its cavity is occluded by a thrombus, the blood current does not pass through the narrowed orifice with sufficient force to produce a fluid vein; in these cases the murmur is absent.

It is, in fact, common to meet with extreme constriction of the mitral orifice on the *post-mortem* table, which was not accompanied by any presystolic murmur during life (I mean of course during the later stages of the case). Indeed Dr Hilton Fagge goes so far as to say, 'my impression is that in the large majority of the cases in which mitral stenosis is found after death, there is no record of the presence of a presystolic murmur during life.'¹

Again, in exceptional cases, the murmur, instead of being presystolic, occurs during the first part of the diastole or long pause, and is separated from the first sound of the heart by a distinct interval. (See fig. 200.) In cases of this description,

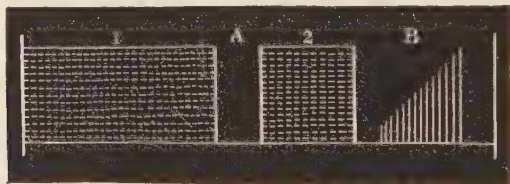


FIG. 200.—Diagrammatic representation of post-diastolic murmur, which occurs in some rare cases of mitral stenosis. It is separated from the commencement of the first sound by an appreciable interval.

the conditions which produce the passage of the blood from the cavity of the left auricle to the cavity of the left ventricle *at the beginning of the ventricular diastole*, must be in excess. Now these conditions are:—(1) the pressure of the blood in the cavity of the left auricle and pulmonary veins; and (2) the suction force which results from the elastic recoil of the left ventricle itself. If either of these forces were in excess, the blood current might pass through the constricted mitral orifice *at the commencement of the ventricular diastole* with

¹ *Russell Reynolds' System of Medicine*, vol. iv. p. 674.

sufficient force to produce an audible fluid vein, which would be heard externally as a diastolic murmur. The first of these conditions, viz., increased blood pressure in the left auricle and pulmonary veins, is probably the chief cause of this form of murmur, though the suction action of the left ventricle cannot be altogether left out of consideration.¹

A *presystolic thrill* can very frequently be felt when the hand is placed over the position of the apex beat.

In addition to the presystolic or diastolic mitral murmur and presystolic mitral thrill, which we may term the *primary* physical signs of mitral stenosis, there are several other physical signs indicative of the altered condition of the mitral segments and of the secondary changes in the physical condition and mode of action of the heart. The most important of these *secondary* physical signs, as I am in the habit of calling them, are:—

(1) *Alterations in the character of the first sound.*—The first sound is usually short and sharp, and rather resembles the normal second than the normal first sound. In some cases, it has a slight thumping character, which Dr George Balfour believes 'is quite pathognomonic (of mitral stenosis) when duly recognised by a practised ear, being simply the last portion of the murmur still extant, the *b* of the murmur as vocalised *rrrrrb*, all the R's being expunged.'² In others, the first sound is replaced by a murmur. This is a common condition, mitral regurgitation being present as we have previously seen, in a considerable proportion of the cases of mitral stenosis. In those cases of combined stenosis and incompetence in which the stenosis is slight, there is only a systolic murmur indicative of regurgitation. In other cases, where the stenosis is more considerable, both presystolic and systolic mitral murmurs are present. In others again, a presystolic murmur is present, but no systolic murmur.

¹ When this chapter was written I had not read Dr Galabin's instructive paper on the Cardiograph in Guy's Hospital Reports, 1875, p. 261. In that paper he very clearly points out the causation of murmurs of this description, and also states that Dr Fagge explained their occurrence by the auricular contraction occurring immediately *after*, instead of immediately *before*, the ventricular systole.

² *Diseases of Heart*, p. 139.

(2) *Reduplication of the second sound.*—This condition, which is diagrammatically shown in fig. 201, is probably present in at least one-third of all the cases of mitral stenosis, and is of considerable diagnostic value. The different views which have been advanced to explain its production, need not be again detailed. (See p. 162.)

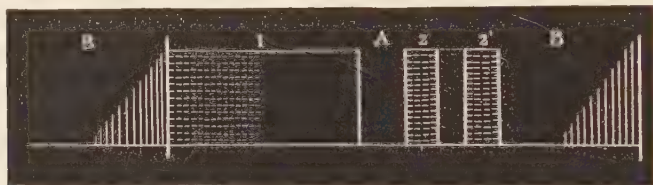


FIG. 201.—Diagrammatic representation of the murmur and reduplicated second sound in mitral stenosis.

(3) *Accentuation of the pulmonary second sound.*—This is a most important physical sign in cases of mitral stenosis, for it is to some extent a measure or gauge of the extent of the mitral lesion. But this point will be more appropriately considered under the prognosis.

(4) *Physical signs which are due to alterations in the size and shape of the heart.*—The *left apex* is usually ill defined and indistinct, but is not displaced downwards and outwards as it is in mitral regurgitation; an exception, of course, occurs in those cases of mitral stenosis in which the left ventricle is hypertrophied either as the result of intrinsic or extrinsic causes; cases of this description are however rare. Dulness, or rather an impaired percussion note is sometimes present over the area of the dilated left auricle, viz., in the second and third left interspaces; it is, I think generally derived from the dilated conus arteriosus of the right ventricle and from the dilated pulmonary artery, rather than from the dilated left auricle, which is more deeply situated in the chest.

Pulsation can also in some cases be felt in the second and third left interspaces. Some authorities believe that this pulsation is derived from the dilated left auricle, or from its dilated appendix; a more probable view is that, I think, which supposes that it is produced by the dilated pulmonary artery

or dilated and displaced conus arteriosus of the right ventricle.

Hypertrophy and dilatation of the right ventricle, tricuspid incompetence, and dilatation of the right auricle, are of frequent occurrence in the later stages of mitral stenosis; and give rise to increased dulness, and other physical signs, which I shall afterwards describe more in detail.

(5) *The characters of the cardiographic tracing.*—In many cases of mitral stenosis the cardiograph affords very important information. The chief alterations are as follows :—

(a) The duration of the diastolic portion of the tracing is prolonged in consequence of the fact that the over-distended left auricle requires a longer time than usual to discharge its contents into the cavity of the left ventricle. (See figs. 202, 203, 204, 205.)

(b) When the stenosis is not very great, there is usually a very marked rise immediately after the wave *k*, which indicates the diastolic relaxation of the ventricle, in consequence either of the fact that the tension of the blood in the left auricle and pulmonary veins is greater than normal, and the blood flow from this auricle to the ventricle, at the commencement of the ventricular diastole, is more forcible than in health; or, as some have supposed, that the auricular contraction commences earlier than usual. (See figs. 202, 203, 204.)



FIG. 202.—*Cardiographic tracing in a case of mitral stenosis.*—(After Galabin.)

‘ Henry A., æt 8. Systolic and long, harsh presystolic murmurs at the apex, both accompanied by thrill. The presystolic murmur commenced immediately from the second sound, and was separated by a short pause from the systole. The bracket in the figure represents the duration of the murmur, which is separated by a distinct interval from the ventricular systole. The letter *a*, indicates the greatest auricular contraction.’¹—(*Guy’s Hospital Reports*, 1875, p. 314.)

¹ For the exact significance of the other letters and parts of the tracing, the reader is referred to the appendix.

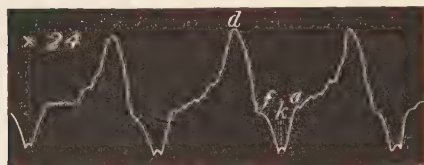


FIG. 203.—Cardiographic tracing in a case of mitral stenosis.—(After Galabin.)

‘George M., æt. 19. Long, loud, and harsh presystolic murmur, commencing immediately from the second sound and running up to the first sound. Pulse 60. The letter *a*, indicates the probable commencement of the auricular contraction.’—(*Guy’s Hospital Reports*, 1875, p. 314.)

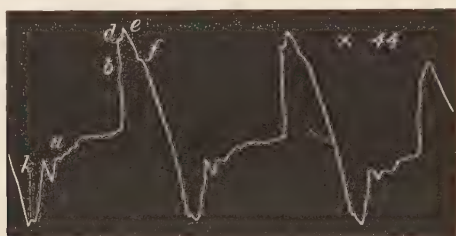


FIG. 204.—Cardiographic tracing in a case of mitral stenosis.—(After Galabin.)

‘Matilda A., æt. 37. Long, rough, presystolic murmur, commencing immediately from the second sound, and leading up to the first sound. Pulse 57.’—(*Guy’s Hospital Reports*, 1875, p. 314.)

(*c*) In those cases in which the stenosis is not great, and in which the muscular wall of the left auricle is hypertrophied, the wave *a*, which indicates the auricular contraction, is increased in height and its base widened, in consequence of the fact that the contraction of the hypertrophied auricle is more forcible and lasts longer than under normal circumstances. (See fig. 202.) In some cases the auricular wave, instead of immediately preceding the rise, which marks the commencement of the ventricular systole, occurs in the earlier part of the diastolic portion of the tracing, owing to the fact that the rhythm of the auricular contractions is altered. (See fig. 203.) When the stenosis is great, the enlargement of the auricular wave is not observed, for the auricular contraction (even when the auricular wall is hypertrophied, and it is often dilated and weak rather than hypertrophied in cases of this description) is unable to propel a sufficiently large quantity of blood through

the narrow orifice to produce a distinct wave in the cardiographic tracing. When the auricular wall is dilated rather than hypertrophied, the size of the auricular wave is diminished rather than increased, whatever be the condition of the mitral orifice. The auricular wave, then, will only be exaggerated in those cases of mitral stenosis in which the auricular wall is capable of contracting powerfully, and in which the stenosis is not too great to prevent this effect of the auricular contraction being manifested in the cardiographic tracing.

(*d*) The diastolic portion of the tracing is in some cases interrupted by a series of elevations and depressions, indicative of the vibrations which are heard as a murmur or felt as a thrill in the mitral area. (See figs. 202, 203, and 204.) These vibrations are usually presystolic, but in some cases they are separated by a distinct interval from the up-stroke, which represents the commencement of the ventricular systole. (See fig. 202.)

(*e*) The systolic portion of the tracing does not, as a rule, present any special features. When the stenosis is considerable, or the left ventricle dilated, the initial rise which marks the commencement of the ventricular systole is followed by a more rapid fall than in health. (See figs. 202, 203, and 204.) In other cases, more especially when the left ventricle is powerful or hypertrophied, the systolic portion of the tracing is sustained and broad. (See fig. 204.)

(*f*) The irregularity in the sequence of the ventricular contractions, which is best demonstrated by means of the sphygmograph, is also seen in cardiographic tracings. (See fig. 204.) The cardiograph also seems to show that in

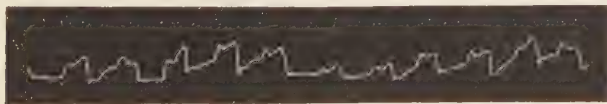


FIG. 205.—Cardiographic tracing in a case of mitral stenosis.—(After Sansom.)

The tracing was taken by Pond's cardiograph. A loud, rolling, or bubbling presystolic murmur at the apex: the irregularity of successive ventricular contractions is well seen.—*Diagnosis of Diseases of the Heart*, p. 271.

some cases the normal sequence of the cardiac contractions is so altered that some of the auricular contractions are not followed by ventricular contractions, as is normally the case.

(6) *Alterations in the character of the pulse.*—The character of the pulse in mitral stenosis depends upon the degree of constriction and the condition of the muscular tissue of the heart, more especially of the left auricle.

When the stenosis is great, the pulse is *smaller* than normal in consequence of the fact that the left ventricle has less blood to expel into the arterial system than under ordinary circumstances. So long as the left auricle is able to empty itself, *i.e.* so long as hypertrophy predominates over dilatation, the pulse may be of good volume, good tension, and perfectly regular, but it usually happens that in consequence of over distention the muscular tissue of the left auricle is every now and again stimulated to premature contraction, which passing to the muscular tissue of the ventricle, is manifested at the wrist in the form of an imperfect pulsation—a small pulse wave, as it were, interposed between two normal beats. (See fig. 206.) The small imperfect pulse wave seems to occur during the down-stroke

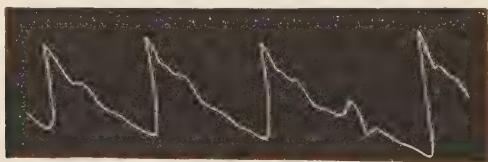


FIG. 206.—*Pulse tracing in Mitral Stenosis.*

The compensation is still fairly good, a second small pulse wave is seen in the down stroke of the third beat.

of the previous beat. In the later periods of the case, and especially when the cavity of the left auricle is constantly over-distended and its muscular fibre in a condition of irritable weakness, the pulse becomes quick and extremely irregular, the irritable muscle discharging, as it were, at rapid

and irregular intervals under the continuous stimulus to which it is subjected in consequence of the increased blood pressure within the auricular cavity.

Diagnosis.—When a case of suspected mitral stenosis comes before us we must endeavour to determine:—

(1) Is mitral stenosis actually present?

(2) If mitral stenosis is present, what is the exact extent and severity of the lesion?

Step. No. 1.—*Is mitral stenosis actually present?*

It is often impossible, as I have previously pointed out, to distinguish slight degrees of constriction of the mitral orifice during life. In cases of this description, there is no distinctive evidence of stenosis; the mitral valve is generally incompetent as well as slightly constricted, and the case presents the usual features of mitral regurgitation. In some cases of this description the cardiograph will probably afford, as Dr Sansom has suggested, important information; when stenosis is combined with incompetence the diastolic portion of the cardiographic tracing is more prolonged than in those cases in which regurgitation is the only lesion.

Passing to those cases in which the constriction is more considerable, we find, that in some the diagnosis is extremely easy, in others difficult.

In those cases in which a presystolic murmur, having its point of differential maximum intensity in the mitral area, *and presenting the rough rolling character* previously described, is present, the diagnosis is self-evident. Such a murmur, so far as my observation goes, is pathognomonic of mitral stenosis.¹

The murmur is not, however, always typical; in some cases, for example, it is not presystolic, but occurs at the commencement of the diastole, and is separated from the ventricular systole by a distinct interval; in other cases it is absent altogether. In some cases, too, it is simulated by the murmur of aortic regurgitation. This is most likely to be the case, as Dr Sansom points out, when the murmur of aortic

¹ It must not, however, be forgotten that Dr Austin Flint holds a different view.

regurgitation is conducted towards the apex, and especially, as is sometimes the case, when it is heard only in the mitral area. 'Cases have been recorded,' says Dr Sansom, 'in which a presystolic murmur has been noted during life, and the autopsy has demonstrated not mitral stenosis but aortic regurgitation.'¹

We must not, therefore, rely upon the mere rhythm of the murmur alone, but must base our diagnosis, as we should always base it, on *all the facts* of the case. We must observe the sound characters of the murmur, the shape and outline of the heart as a whole and of its component parts, the condition of the pulmonary second sound, and the exact characters of the pulse. If these points are accurately noted, there should be no difficulty in distinguishing aortic incompetence from mitral stenosis, even in those cases in which the murmur is only heard at the apex, and is more or less presystolic; for the secondary alterations in the heart and circulation, which result from aortic incompetence, are totally different from those caused by mitral constriction. (See pp. 480 and 505.)

Those cases in which the murmur is wanting altogether, present greater difficulties. A patient, for example, comes under observation suffering from dropsy of the feet and lower extremities, shortness of breath and cough; he is markedly cyanotic, the right heart enlarged, the tricuspid valve perhaps incompetent, the second sound reduplicated, the pulmonary second sound markedly accentuated, the pulse quick, small, weak, and irregular. There is no murmur in the mitral area, the left apex is ill defined, but is not displaced outwards and to the left. The lungs are markedly emphysematous, the normal respiratory sounds are replaced by bronchitic râles.

Bronchitis and emphysema, with extensive secondary changes in the right heart, are evidently present, but it may be extremely difficult or even impossible to decide whether these conditions are primary or secondary to a constriction of the mitral orifice. It is only by careful attention to the history of the case that the point can be decided

¹ *Lettsomian Lectures*, p. 70.

with any approach to certainty. A history of rheumatism, especially of articular rheumatism; of shortness of breath on exertion, the dyspnœa having gradually and steadily increased, and being present on exertion, whether the patient was suffering from 'cold' (bronchitis) or not, is suggestive of mitral constriction; *vice versâ* when there is no rheumatic history, when the patient has suffered from repeated attacks of bronchitis, when the shortness of breath has not steadily and gradually increased, and especially if there have been intervals (between the attacks of bronchitis) in which his 'wind' was pretty good, the probability of primary lung disease and the absence of mitral stenosis are indicated.¹

In some cases of cirrhosis of the lung with secondary hypertrophy and dilatation of the right heart, the same difficulty in diagnosis occurs.

In those cases of advanced mitral disease in which there is no murmur, and in which pulmonary complications (bronchitis, emphysema, cirrhosis, etc.) are present in small degree or altogether wanting, the diagnosis can be made with much greater facility. In cases of this description the accentuated pulmonary second sound and the hypertrophied, or hypertrophied and dilated right heart show, that there is some obstruction to the passage of the blood through the lungs; and if there is no evidence of primary lung disease (emphysema, cirrhosis, etc.) sufficient to produce the obstruction, we may with some confidence conclude that the cause of the obstruction is placed at the mitral orifice.

Careful cardiographic observations would probably help us in distinguishing these cases. The character of the pulse too, is, I think, of some importance. In advanced mitral constriction the pulse is more likely to be irregular than it is in bronchitis and emphysema and in the other primary lung affections in which the difficulty in diagnosis, which we are now considering, is likely to occur.

Cases, in which hæmoptysis occurs as an early symptom,

¹ Cases of this description, in which the attacks of bronchitis date back from childhood, are often attended with extreme cyanosis, and are likely to be mistaken for cases of congenital malformation of the heart.

may be mistaken for commencing phthisis. I remember making a mistake of this kind soon after commencing practice, which I have never forgotten. A young man consulted me for hæmoptysis ; he was thin and spare ; some bronchial râles and a well-marked presystolic mitral murmur, were present. The breathing at the right apex was a little harsher than at the left (a condition which is, of course, quite compatible with perfect health). I erroneously attributed the hæmoptysis to primary lung disease, an opinion which was shortly afterwards very properly set aside by the late Dr Murchison. The patient is, I believe, still alive, and has never had any symptoms or signs of phthisis. Cases in which the same mistake is committed are by no means uncommon, and more than one has since come under my own personal observation.

Step No. 2. If the case is one of mitral stenosis, what is the extent and gravity of the lesion ?

This step in the diagnosis will be more appropriately considered under the prognosis, to which I now pass.

Prognosis.—Having decided that the case is one of mitral stenosis, we must endeavour to determine what is the gravity of the lesion. Here, as in the case of mitral regurgitation, the extent and gravity of the lesion are not altogether synonymous terms. The gravity of the lesion is, in short, determined by observing :—

1. The degree or amount of the stenosis. We form an opinion on this point by showing—(a) the extent of the secondary changes produced in the heart itself ; and (b) the amount of mechanical derangement produced in the arterial and venous systems respectively. The extent of the hypertrophy and dilatation of the right heart is the most important guide to the extent of the mitral lesion. The degree of accentuation of the pulmonary second sound is also of great value, more especially in the earlier stages of the case. In attempting to gauge the extent of the constriction by the loudness of the pulmonary second sound, it is important to take into account the condition of the lungs on the one hand, and of the right ventricle on the other. Accentuation

of the pulmonary second sound is only, of course, an indication of increased blood-pressure in the pulmonary artery; and since increased pulmonary tension may be due to pulmonary causes (such as bronchitis and emphysema), the exact condition of the lungs must be accurately determined before we can attempt to measure the degree of the mitral stenosis by the extent of the accentuation of the pulmonary second sound which is present. Again, it is essential to remember, that the degree of the pulmonary tension, and therefore the loudness of the pulmonary second sound, depend upon the condition of the right ventricle. With the same amount of obstruction in front, the pulmonary second sound will be much louder when the ventricle is hypertrophied and acting powerfully than when it is dilated and acting feebly. The mere loudness of the pulmonary second sound is not, therefore, any accurate guide to the extent of the mitral constriction, unless we at the same time take into account the condition of the lungs and of the right heart.

2. The capabilities of compensation and the powers of resistance possessed by the particular patient under observation.

3. Whether the lesion of the mitral valve is progressive or stationary.

4. The presence or absence of complications.

The same remarks which have previously been made with regard to headings 2, 3, and 4, in speaking of the prognosis of mitral regurgitation apply here. (See page 459, *et seq.*)

Treatment.—In treating cases of mitral stenosis, the indications are the same, and the methods of treatment are similar to those which have been described as suitable in cases of mitral regurgitation. (See page 467.) I need not again enter into details, but must once more emphasise the statement, that in the earlier stages, and so long as compensation is perfect, little or no drug treatment is required.

AORTIC INCOMPETENCE.

Definition.—Aortic incompetence includes all those conditions which interfere with the perfect closure of the aortic valve, and allow a regurgitant current to pass from the aorta into the cavity of the left ventricle during the ventricular diastole.

Ætiology and Pathology.—Aortic regurgitation is a common condition, though not nearly so common as mitral regurgitation. It is comparatively seldom met with in young persons, but is of most frequent occurrence during active manhood and the later periods of life. It is much more common in men than in women, a circumstance which is explained by the fact that the root of the aorta and aortic valve segments are more subjected to strain, and are more liable to be affected with atheroma and chronic inflammation in the male than in the female.

In order to comprehend intelligently the diseases of the aortic valve, it is essential to remember that it (the valve) forms a barrier between the arterial system, on the one hand, and the heart, on the other; and that the morbid processes which produced lesions of the aortic segments arise in some cases in the base of the aorta, in others, in the endocardial lining membrane of the heart. Further, it is all important to bear in mind, that the force with which the aortic valves are closed, depends upon the condition of the aortic, *i.e.* of the systemic arterial blood-pressure. But in order that these points may be thoroughly understood, I must now describe in detail the anatomical construction of the aortic segments, and the exact manner in which their closure is effected.

The aortic orifice is circular in form, and the aortic valve consists of three semi-lunar flaps; each flap is attached by its convex border to the side of the artery at the place where it joins the ventricle, whilst its other border, which is nearly straight, is free, and projects into the interior of the vessel. The segments of the valve are composed of fibrous tissue, covered by a prolongation of the endocardium on the one side, and of the inner coat of the aorta, on the other. The

thickness of the segments varies in different parts. A tendinous band strengthens the free edge of the flap, and at the middle of the free edge there is a slight fibro-cartilaginous thickening, the *nodulus* or *corpus Arantii*. Other tendinous fibres, arising from the attached border, run in the valve towards the nodule, occupying its whole extent, except two narrow lunulated portions, one on each side adjoining the free margin of the valve. These parts, which are named *lunulæ*, are therefore thinner than the rest. There is also a strengthening fibrous cord surrounding the attached border of each valve. The wall of the aorta is bulged out opposite each semi-lunar flap; these bulgings are known as the *sinuses of Valsalva*. (See fig. 207.) One of the sinuses is situated

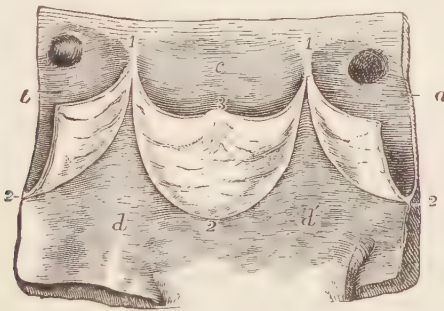


FIG. 207.—*Portion of the aorta and wall of the left ventricle with one entire segment and two half segments of the aortic valve.*—(After Quain.)

a, b, c, sinuses of Valsalva opposite the segments; in *a*, and *b*, the apertures of the coronary arteries are seen; *d, d'*, the inner surface of the wall of the ventricle; *1, 2*, curved attached border of the segments; *3*, the middle of the free border (*corpus Arantii*).

anteriorly, the other two posteriorly. The right coronary artery arises from the anterior sinus, and the left coronary artery from the left posterior sinus. The right posterior sinus is sometimes called the *intercoronary sinus*.¹

The action of the valve is as follows:—when the left ventricle contracts the valve segments are burst open, but do not

¹ *Quain's Anatomy*, vol. ii. p. 490.

come into immediate contact with the aortic wall, as was at one time supposed; they seem to be retained (probably as the result of reflux currents passing round the root of the aorta and into the sinuses of Valsalva) in an intermediate position; the orifice of the valve, when it is opened, is more or less triangular in shape. As soon as the contraction of the left ventricle ceases, the pressure behind the valve becomes negative;¹ reflux currents at the root of the aorta are produced, which float the segments of the valve into close apposition. The elastic recoil of the aorta then occurs, the root of the aorta including, of course, the sinuses of Valsalva, is bulged out, the valve flaps are tensely stretched, and are brought into firm and accurate contact. It must also be remembered that the strain of this forcible closure is borne by the *corpora Arantii* and tough fibrous portions of the valve flaps, and that the delicate *lunulæ* are not subjected to any strain, but are simply pressed against each other, so as most effectually and completely to close the orifice.

Now aortic incompetence may be due either to:—

1. Alterations of the valve segments, which prevent their perfect adaptation and closure;

2. Dilatation of the base of the aorta, the valve segments being healthy. This, which is a rare condition, is termed *relative* incompetence. Let us now shortly consider each of the pathological causes of these two forms in detail.

Alterations in the valve segments, which prevent their perfect adaptation and closure, may be due to:—

1. *Congenital malformation, or disease in intra-uterine life.*—This is an extremely rare cause of aortic incompetence, though congenital malformation of the valve segments is not uncommon. In some cases of congenital malformation there

¹ Foster states that 'when in a closed channel a rapid current suddenly ceases, a negative pressure makes its appearance in rear of the fluid, and sets up a reflux current. So when the last portions of blood leave the ventricle a negative pressure makes its appearance behind them in the ventricle, and leads to a reflux current from the aorta towards the ventricle.'—(*Text-Book of Physiology*, p. 143.) The elastic recoil of the ventricle produces also, as we have previously seen, considerable negative pressure in the ventricular cavity, which must contribute very materially to the production of these reflux currents.

are only two segments, in others more than three, but in the majority of cases of this description the valve is competent, and it is only by accident the condition is discovered after death. It must, however, be remembered that malformed valves seem more liable to be attacked by endocarditis in later life than naturally formed ones. In other cases the number of segments is normal, but adjacent flaps are adherent at their edges. In some cases of this description, in which the valve has become incompetent in later life, it has been supposed that the adhesions were due to intra-uterine disease.

2. *Traumatic rupture of the valve flaps.*—Rupture of the aortic segments, as the result of traumatic injury or sudden effort, is extremely rare in perfectly healthy individuals. It is probable that in many cases in which a valve flap has given way under sudden effort or strain, it was weakened by previous disease, which had not advanced sufficiently far to give rise to symptoms. Rupture, the result of ulcerative inflammation, is of course more common. (See figs. 168 and 169.) In both forms of rupture the regurgitation is usually very free; cases of this description generally run a rapid course, and, so far as we know, are invariably fatal. In the following case, in which the rupture was possibly partly due to strain, but chiefly to ulceration, the patient survived for a considerable time :—

Case.—J. R., æt. 36 (height 5 ft. 9½ in., weight when in health, 12 st. 12 lbs.), an extremely powerful man, was admitted to the Newcastle-on-Tyne Infirmary under my care on November 12th 1874.

The *history* which he gave of the commencement of the case, was interesting, and was as follows :—He stated that with the exception of a cough, from which he had suffered for some two years before his present illness commenced, he had enjoyed excellent health, never having been in bed from illness for a single day. One morning at the end of June 1874, he noticed a peculiar noise on getting out of bed in the morning; he thought it proceeded from under the bed, but failing to find any cause for it there, and after searching the other parts of his house and finding that it always accompanied him, he concluded that it originated inside his own body. On consulting a doctor he was told it proceeded from his heart. Before the noise commenced, he had been engaged on ‘a very heavy job lifting stones,’ but was not aware that he had strained himself. For some weeks after the noise was first noticed, he was able to continue

his ordinary employment—that of a labourer. He then caught cold and became short of breath. The shortness of breath and cough gradually increased, his legs swelled, and he was obliged to apply for admission to the Infirmary. The cardiac murmur, for such it proved to be, continued auto-audible for a few weeks, and could be heard at a distance of two feet from his body by bystanders; it then ceased, and he has not heard it since.

On *examination*, cardiac dropsy and the other signs of systemic venous engorgement were present; the apex beat was in the sixth interspace, two inches below the left nipple; the superficial vessels presented the characteristic Corrigan pulsation of aortic incompetence; the area of præcordial dulness was much increased; a faint systolic thrill could be felt in the third left interspace just outside the sternum; a double bellows murmur was loudly heard at the base of the heart, the diastolic portion of the murmur was propagated over the course of the aorta and down the sternum; at the lower end of that bone it was loudly heard; it could also be heard, but only indistinctly, at the cardiac apex. An independent systolic mitral murmur was also present. There was extensive bronchitis.

Under digitalis the patient greatly improved, and was made an outpatient on November 27th.

On January 4th he was re-admitted very much worse; and he died on January 24th, the dropsy and orthopnoea having rapidly increased notwithstanding the free administration of digitalis and other remedies.

On *post-mortem* examination the aortic valve was found to be extremely incompetent, the left posterior cusp was completely ulcerated through, a nipple-like process, the edges of which were perfectly smooth (see fig. 208) being all that remained; the surface of the aorta corresponding to the affected valve was markedly atheromatous, the adjacent portions of the vessel being comparatively healthy; the left ventricle was enormously hypertrophied and dilated; the mitral and tricuspid orifices dilated; the heart weighed 45 ounces. The lungs contained several recent patches of pulmonary apoplexy; there was great subcutaneous dropsy and considerable effusion into all the serous cavities.

The case is interesting from many points of view; but space does not allow me to go into details further than to say, that it seems completely to disprove Dr B. Foster's theory, that when the left posterior valve segment is incompetent, the murmur, instead of being conducted down the sternum is propagated towards the apex of the heart. The great weight of the heart must also be noted.

3. *Acute Endocarditis*.—Aortic incompetence is comparatively rarely established during the acute stage of simple endocarditis; in the ulcerative form of endocarditis it is of



FIG. 208. *Ulceration of one of the Aortic Cusps. (Natural size.)*

The aortic segment corresponding to the posterior coronary artery is completely ulcerated through, a nipple-like process *a*, the edge of which is perfectly smooth being all that remains; the sinus of Valsalva corresponding to the affected cusp is dilated, and that portion of the aorta (*b*), which is situated above the affected valve is atheromatous, the other portions of the vessel (*c*), being comparatively healthy.

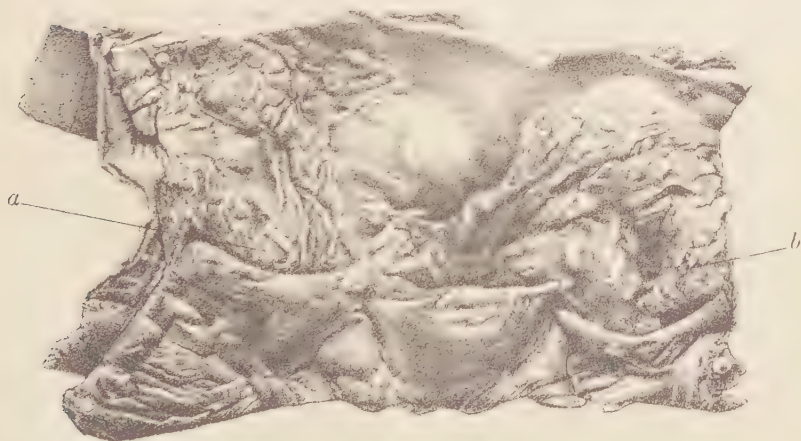


FIG. 209. *Atheroma of the base of the Aorta: Obstruction of the orifices of the Coronary Arteries; Extreme incompetence of the Aortic Valve. (Natural size.)*

The base of the aorta is markedly atheromatous, and contains several large, flat, smooth calcareous plates; the orifice of one of the coronary arteries (*a*), is completely occluded, while that of the other (*b*) is very much narrowed; the segments of the aortic valve are thickened and crumpled; the valve was extremely incompetent.

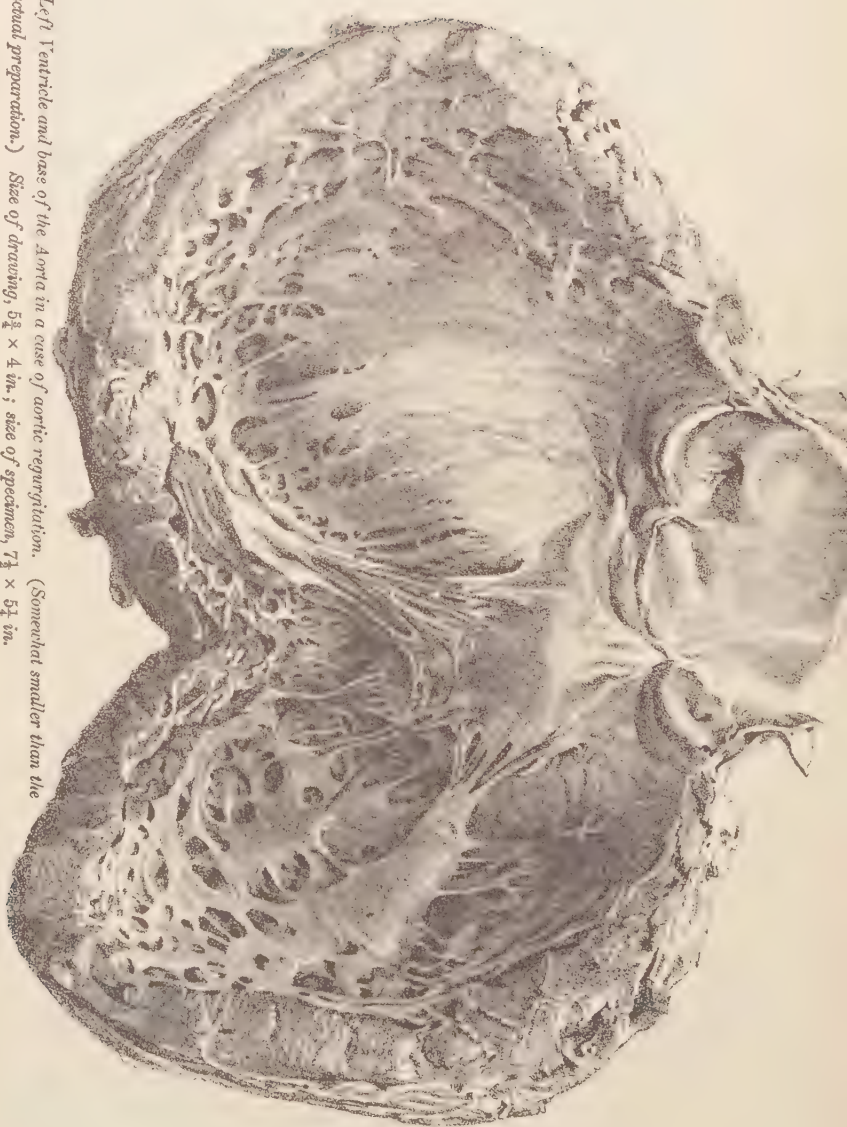


FIG. 210.

The interior of the Left Ventricle and base of the Aorta in a case of aortic regurgitation. (Somewhat smaller than the actual preparation.) Size of drawing, $5\frac{3}{4} \times 4$ in.; size of specimen, $7\frac{1}{2} \times 5\frac{1}{4}$ in.

The aortic cusps are curled inwards and shrunken, giving rise to very free regurgitation; the cavity of the left ventricle is markedly dilated, there is also considerable hypertrophy.

frequent occurrence.¹ The appearances which the valve presents in cases of acute, simple, and ulcerative endocarditis, have previously been fully described. (See page 403.)

4. *Chronic Endocarditis.*—This is a very common cause of aortic incompetence. I need not again detail the different causes of chronic endocarditis, but I may say that, in the majority of cases of aortic incompetence which result from chronic endocarditis (as distinct from atheroma), the inflammation of the endocardium is either rheumatic or the direct result of strain. In all the large centres of laborious work, in Leeds, for example, as Dr Clifford Allbutt has pointed out, and in Newcastle, as I know from personal experience, cases of aortic incompetence are frequently met with in young men who follow laborious occupations, necessitating sudden and great variations in the arterial pressure, and who have never suffered from rheumatism, syphilis, gout, or the other diseases with which endocarditis and atheroma are usually associated. In cases of this description the base of the aorta is usually dilated; the regurgitation is, in fact, usually due both to organic changes in the valve segments and to relative incompetence.

The alterations which are produced in the aortic valves by chronic inflammation are various. In the great majority of cases, all three segments are involved, though in many cases the different segments are unequally implicated. The valve segments are thickened; they are often of a cartilaginous consistency, and in many cases infiltrated with calcareous deposits. The natural elasticity of the affected segments is impaired or altogether destroyed. The thickening more particularly involves the free edges of the flaps, the *lunulæ* being in many cases completely obliterated. In many cases, the whole of the affected segment is contracted and shrunk; in others, the free margins are crumpled and turned inwards towards the base of the aorta (see figs. 209 and 210); in

¹ I do not of course mean to say that aortic regurgitation, the result of ulcerative endocarditis is frequently met with in practice, but that, *when* the endocardium is attacked by the ulcerative form of endocarditis, aortic regurgitation frequently occurs. Cases of ulcerative endocarditis are comparatively rare.

others, again, the free edge is inverted towards the ventricle. In a few cases, one or more of the valve segments have appeared to me to be more voluminous and dilated than natural. In many cases the adjacent segments are adherent at their edges, and masses of calcareous vegetations are not unfrequently found at the points of adhesion. It occasionally happens, that the partition formed by this process of adhesion between two adjacent flaps breaks down and gives way, two adjacent pouches being, as it were, thrown into one.

5. *Atheroma*.—Atheromatous changes at the base of the aorta are very frequently associated with aortic incompetence; in some cases the valvular lesion is identical with that which is produced by chronic endocarditis (chronic rheumatic endocarditis); cases of this description may occur in young subjects, the valvular lesion and the atheroma usually depending upon a common cause, viz., strain; in other cases, and these are more apt to occur in old people, the valvular lesion is rather degenerative than inflammatory in character, the valve segments being calcified, but their form being comparatively little interfered with. (Atheroma of the root of the aorta does not necessarily produce disease of the aortic valves. Cases are occasionally met with, in which the whole base is extremely atheromatous, but in which the valve segments are perfectly natural and elastic.) In the atheromatous disease of the aortic segments which occurs in old people, marked stenosis is often combined with regurgitation; and in all cases of aortic incompetence, in which the regurgitation depends upon organic changes in the valve segments, some stenosis may be present. In many cases of aortic incompetence, recent vegetations and coagula are found on the affected segments, chronic disease of the valve segments being, as we have previously seen, a powerful predisposing cause of recent endocarditis.

Method of testing the aortic valve after death.—The heart is to be removed in the usual manner, the aorta and pulmonary artery being divided about two inches above their valvular orifices. The cavity of the left ventricle must then be opened, care being taken to avoid division of any of the larger branches

of the coronary artery. Any clots in the ventricular cavity, and especially any clots which are sticking in the aortic orifice must then be gently removed with the finger, and a stream of water allowed to flow from above into the aorta and pulmonary artery. If the valve holds water, it is, of course, quite competent. It must, however, be remembered, that it is difficult to fulfil all the conditions which are present during life, more especially the *forcible* distention of the base of the aorta; hence it is that in some cases in which the valve seemed perfectly healthy during life, water slowly makes its way through into the ventricle after death.

Incompetence due to dilatation of the base of the aorta, the valve segments being healthy. Relative incompetence of the aortic valve is rare, but that it does sometimes occur I am perfectly satisfied from cases which have come under my own personal observation. I have seen several cases in which the base of the aorta was dilated (either a uniform dilatation due to strain and associated with atheroma, or localised (aneurismal) dilatation immediately above the valves), the valve segments quite healthy, but the valve incompetent to the water test after death, and in which there were all the usual characteristic signs of aortic regurgitation during life.

Pathological Physiology.—The *first* effect of aortic regurgitation is, of course, to allow some of the blood which ought to be retained in the aorta to flow back into the cavity of the left ventricle; in other words, to produce anæmia in the arterial system, and more or less engorgement behind the valvular orifice, *i.e.* in the cavity of the left ventricle; so long as the mitral valve is competent, the circulation behind the mitral orifice is very little interfered with.

The *second* effect is to produce a series of changes in the heart and circulation, which are briefly as follows:—

Effect on the heart.—In aortic regurgitation the cavity of the left ventricle receives blood from two sources, *viz.*, the aorta and the left auricle; it is therefore more rapidly and more fully distended than in health. In consequence of the forcible distention of its flaccid walls *dilatation* is produced

(see figs. 171 and 210), while its muscular wall is, by reason of the rapidity with which the cavity is filled, more rapidly stimulated than in health, and the *frequency of the cardiac contractions is increased*. The increased effort which is required to expel the extra quantity of blood which the cavity contains leads also to the production of *hypertrophy*.¹

Now all of these changes—dilatation and hypertrophy of the ventricle, and increased rapidity of the cardiac contractions—are within certain limits beneficial and salutary. When, for example, the dilatation is just sufficient to accommodate the quantity of blood which regurgitates from the aorta, and at the same time to receive the normal quantity of blood from the left auricle, and when the hypertrophy is sufficiently great to enable the cavity to be completely emptied at each systole, the compensation is perfect. Under such circumstances the circulation behind the mitral valve is little if at all interfered with; and provided that the anæmia in the arterial system, which necessarily occurs during the ventricular diastole (*i.e.* in consequence of the regurgitation of some of the blood which ought to be strained in the arterial system into the left ventricle) is not great, and provided that disastrous consequences are not produced in the peripheral arteries as the result of their excessive distention during the ventricular systole, there are few, if any, symptoms.

In many cases of aortic regurgitation in which the leak is a slight one, in which the valvular lesion is stationary, and in which the heart and organism as a whole are healthy, satisfactory compensation is maintained for years, the patient being able to lead a comfortable and active existence.

In other cases in which the leak is greater, and more especially in those cases in which the valvular lesion is progressive,

¹ The heart is heavier in aortic regurgitation than in any other condition. Dr Hilton Fagge has recorded a case in which it weighed 48 ounces, and I myself have met with a heart weighing 45 ounces. (See case p. 501.) It is important to observe, as was first pointed out by Traube (quoted by Rosenstein in *Ziemssen's Cyclopædia*, vol. vi. p. 134), 'that the papillary muscles are not proportionately enlarged with the other parts of the left ventricle—that is, they are not round and hypertrophied, but generally lengthened and flattened, corresponding to the considerable strain to which they are exposed during diastole.'

or in which the heart and organism as a whole are in an unhealthy condition, the compensation which for a time perhaps was sufficient to balance the lesion ultimately gives way and disastrous consequences result. In cases of this description, dilatation gradually gains the upper hand, and the arterial anæmia becomes excessive. In consequence of the dilatation of the ventricle, various important results may ensue; in some cases, the degenerated and weakened muscle is paralysed, as it were, by the over-distention of the cavity, the heart's action is arrested in diastole, and the patient suddenly drops dead; in others, the dilatation leads to relative incompetence of the mitral orifice; in others again, the nutrition of the ventricle fails, and a condition of asystole is gradually established.

Effect on the left auricle, pulmonary circulation, right heart, and systemic venous circulation.—In the earlier stages of aortic regurgitation, and throughout those cases in which compensation remains perfect, there is little or no interference with the free passage of the blood from the cavity of the left auricle; the engorgement of the pulmonary and systemic circulation is so slight that it leads to no practical consequences. In the later stages, when dilatation is in excess of hypertrophy, or when relative incompetence of the mitral valve is established, the pulmonary and systemic circulations may become seriously embarrassed; and the secondary consequences which I have previously described in detail, may of course become established.¹ Mitral regurgitation due to organic changes in the valve segments is not unfrequently associated with aortic regurgitation, the same pathological process (chronic endocarditis or atheroma) which produces the aortic lesion having simultaneously attacked, or subsequently extended to, the anterior segment of the mitral valve, which is continuous with the posterior aortic segments. (See figs. 177 and 181.)

Effects on the arterial circulation.—The effects which aortic incompetence produces on the arterial circulation are very

¹ Relative incompetence would probably be of more frequent occurrence if it were not for the fact that in many cases of aortic incompetence the basal ring of the mitral valve is rigid in consequence of atheromatous changes.

striking. In consequence of the hypertrophied and dilated condition of the left ventricle, the arterial system is very forcibly and very fully distended during the ventricular systole; as a result of the excessive strain, atheroma is apt to be produced and arterial rupture to occur. In consequence of the regurgitation, the blood pressure in the aorta and arteries generally, suddenly falls with the occurrence of the ventricular diastole, and a condition of peripheral anæmia, the extent of which varies of course with the extent of the regurgitation, remains until the occurrence of the next ventricular systole. It will now be readily understood that the increased frequency of the cardiac contractions, which I have already alluded to, is distinctly beneficial and compensatory, for not only does the contraction of the ventricle arrest the reflux, but it, at the same time, distends the arteries, and is, therefore, in a twofold manner beneficial.

The effect which aortic regurgitation produces on the coronary circulation, *i.e.* on the nutritive supply of the heart itself, is a subject of much importance, and has given rise to considerable debate. It was formerly supposed that during the ventricular systole the mouths of the coronary arteries were covered up by the flaps of the aortic valve, and that the blood was pumped into these vessels by the elastic recoil of the aorta, during the diastole of the heart. It was therefore supposed, that in aortic incompetence the distention of the coronary arteries would be imperfect, and the nutrition of the heart seriously impaired. Thanks to the observations of Martin and Sedgwick, we now know that the coronary arteries are distended like all the other arteries of the body during the ventricular systole; while the fact that the left ventricle is able to become so enormously hypertrophied as it does in many cases of aortic incompetence, seems to show that the supply of blood to the heart is, in most cases, amply sufficient. Nevertheless it must, I think, be conceded that free aortic regurgitation must materially interfere with the passage of the blood into the coronary arteries during the ventricular diastole. We may further suppose that this interference will be greatest when the coronary segments of the valve are ruptured, or extensively destroyed by disease.

Clinical history.—The symptoms met with in cases of aortic incompetence are more intelligible when it is remembered—

1. That the arterial system is suddenly and widely distended by the large quantity of blood which is pumped into it by the hypertrophied and dilated left ventricle.

2. That on the cessation of the ventricular systole, a sudden collapse in the arterial tension occurs, and the arteries are rapidly emptied during the ventricular diastole.

3. That in all cases of aortic incompetence, there is a tendency to sudden over-distention and paralysis of the left ventricle.

4. That in a considerable number of cases the peripheral arteries are atheromatous; that in others the thoracic aorta is dilated or is in an aneurismal condition; while in others, the sensory nerve fibres which ramify so abundantly on the outer surface of the aorta, are liable to be irritated and implicated by inflammatory processes originating in the interior of the vessel.

The *onset* is, as a rule, slow and insidious; in exceptional cases, as, for example, when the valve flaps are rapidly destroyed by ulceration, or ruptured as the result of traumatic injury or excessive strain, urgent symptoms may be quickly or suddenly developed.

Chronic cases, which we have now more particularly to consider, may, for practical and teaching purposes, be conveniently divided into two groups:—

1. *Cases in which the lesion is a stationary one, and in which the regurgitation is slight, and insufficient to produce any considerable alteration in the condition of the left ventricle or in the peripheral arterial circulation.*

In cases of this description there are practically no symptoms. The patient may perhaps be a little paler than natural, he may be more easily fatigued, and unequal to prolonged mental effort or bodily exertion, but nevertheless able to go through a large amount of mental work, and to enjoy life, and capable of active exercise. All physicians of large experience are probably acquainted with professional men

who have for years been the subjects of aortic incompetence ; to the eye of a layman they may appear to be perfectly healthy, but the skilled observer can usually detect evidence of their disease in the pallor of the face and the jerking pulsation in the carotids and more particularly in the peripheral vessels. The duration of these cases is usually long (10, 20, 30 years or more). It must, however, be remembered that there is always a risk of the lesion increasing, of the failure of compensation, and of the occurrence of sudden death from temporary over-distention and paralysis of the left ventricle.

2. Cases in which the lesion is more extensive, and in which considerable alterations in the condition of the left ventricle, and in the peripheral arterial circulation are produced.

So long as the compensation is fairly good the symptoms are not prominent. The face is pale, and usually has a somewhat anxious expression. Although the patient can, as a rule, take a considerable amount of active exercise without feeling short of breath, there is a loss of nerve tone and of muscular power and endurance ; pains in the back and lower extremities, such as result from nervous exhaustion are sometimes observed, and in more than one case twitchings in the lower extremities, not unlike the fibrillary twitching of progressive muscular atrophy, have come under my notice. These symptoms depend, I think, upon anæmia of the lumbar enlargement of the spinal cord. Twitchings in the facial muscles may also occur. In one case which came under my observation some years ago, twitchings in the facial and labial muscles, slowness in cerebation, and thickness and hesitation in labial speech, all seemed to depend upon cerebral anæmia, the result of very free aortic regurgitation, and gave the case a close resemblance to general paralysis of the insane. Noises in the ears, flashes of light before the eyes, throbbings of the peripheral blood vessels, headache, giddiness or actual fainting, and many other symptoms (due either to sudden distention of the arterial system during the cardiac systole, or to anæmia during diastole) are of common occurrence. Palpitation is frequently complained of. Pain in the region of the heart,

and in some cases attacks of true angina pectoris are not very uncommon; these symptoms depend, I think, in some cases upon over-distention and spasm of the ventricular wall, in others upon irritation of the branches of the cardiac plexus of nerves.

When the compensation begins to fail, all of these symptoms become more prominent, and the patient now, perhaps, for the first time, is actually short of breath. The arterial anæmia during diastole and the shortness of breath gradually increase. Dropsy and pulmonary complications may now develop, and usually show that the mitral valve has become incompetent. The symptoms, in fact, in the terminal stage of aortic regurgitation are partly due to engorgement of the pulmonary and systemic venous circulations, the result of mitral regurgitation, and partly to arterial anæmia, the result of the primary (*i.e.* the aortic) lesion.

Embolic symptoms, caused by particles of fibrine which have become detached from the aortic segments and carried to distant parts of the circulation, may occur at any stage of the case. In cases of this description, acute or sub-acute inflammatory changes are usually present in addition to the chronic lesion, and it is in consequence of these more active changes that the fibrine is deposited on the aortic segments. When the aorta is much dilated or in an aneurismal condition, other symptoms, which I shall afterwards describe in treating of aneurisms may be present. When the arteries are extensively atherosed, apoplexy and other complications may arise.

The duration of cases of aortic regurgitation included under this group is very variable. Once compensation begins to fail, the downward progress is usually rapid. Death may occur at any stage from sudden failure of the action of the left ventricle.

Physical signs.—A diastolic murmur, having its point of differential maximum intensity at the base of the heart, or (in some cases) at the lower end of the sternum, heard at the aortic cartilage and propagated downwards along the sternum or towards the apex of the heart (see fig. 211), is characteristic

of aortic regurgitation. The murmur is usually soft and blowing; in some cases it is very faint, in others loud and (occasionally though rarely) musical. A systolic aortic murmur, which is usually due to associated stenosis of the aortic orifice, but which may be caused by dilatation of the ascending thoracic aorta, or possibly by anæmia, is frequently also present.

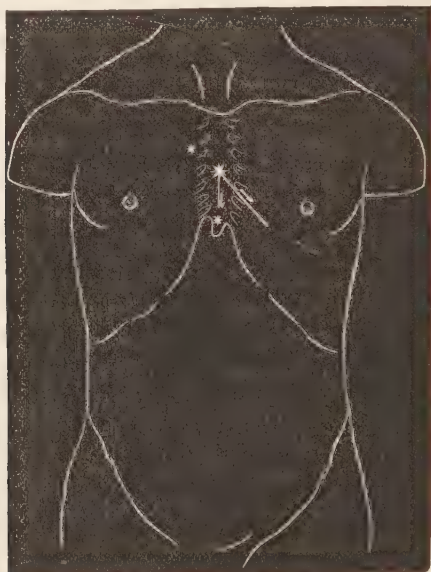


FIG. 211.—Outline figure showing point of differential maximum intensity (*) of the diastolic murmur (aortic regurgitation); and the direction in which it is propagated.

In some cases the second sound is completely replaced by the murmur; in others, both the second sound and the murmur are audible. Occasionally the second sound, which is totally obscured at the base of the heart, can be heard in the carotid artery. (Rosenstein.)¹

A diastolic thrill may sometimes (though rarely) be felt when the hand is placed over the base of the heart. (A systolic thrill is present in many of the cases in which a double—systolic and diastolic—aortic murmur is audible.)

¹ Ziemssen's *Cyclopædia of Medicine*, vol. vi. p. 136.

In addition to the diastolic murmur and diastolic thrill, which we may term the *primary* physical signs of aortic incompetence, others, due to secondary changes in the left ventricle and the altered condition of the arterial circulation, are present. The more important of these *secondary* physical signs are :—

1. Displacement of the apex downwards and outwards.
2. Increase of the cardiac dulness.
3. A powerful heaving action of the heart.—(This condition which depends upon hypertrophy of the left ventricle, may not be present in the later stages of the case, *i.e.* when dilatation is in excess of hypertrophy.)
4. Alterations in the character of the arterial pulse.—The sudden distention of the arterial system which results from the large quantity of blood which is propelled into the aorta by the dilated and hypertrophied left ventricle, and the sudden collapse which occurs as the result of the back flow into the left ventricle during the ventricular diastole, produce very striking alterations in the carotid, radial, and other peripheral pulses.

The pulse is quicker than normal ; as a rule it is quite regular ; the distention of the artery is sudden and the pulse is jerking, visible, collapsing, and tortuous ; it has been called the water-hammer pulse, or Corrigan's pulse (after Sir Dominic Corrigan, who first described these features). The jerking, visible and collapsing characters, are usually made more prominent by raising the arm above the head. It must, however, be remembered that when the heart is acting very feebly, raising the arm may make the pulse less, rather than more visible. A sphygmographic tracing shows that the artery is over-distended during the ventricular systole, and under-distended during the ventricular diastole. The aortic wave is situated low down in the tracing, and is usually, but not invariably, less prominent than in health ; in some cases it is altogether effaced. (See figs. 212 and 213.)

A systolic murmur, which is probably due to vibrations produced in the arterial coats by the rapid distention during the ventricular systole, can often be heard in the peripheral

vessels. When the regurgitation is free, a diastolic murmur can also be heard in the peripheral arteries. When the arte-

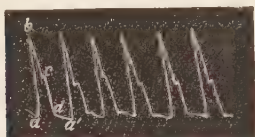


FIG. 212.—Pressure $2\frac{1}{2}$ oz.

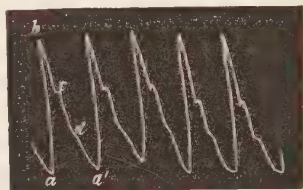


FIG. 213.—Pressure 3 oz.

FIGS. 212 and 213.—Pulse tracings in a case of Aortic Regurgitation.

The tracing shown in fig. 212 was taken on the patient's admission to hospital; the arteries were almost empty during the ventricular diastole. *a-b*=up-stroke; *b*=apex; *c*=tidal wave; *d* indicates the position of the aortic wave, which is absent in this tracing.

FIG. 213.—Taken from the same patient after the administration of digitalis. The letters have the same significance as in fig. 212.

rial walls are atheromatous, when the mitral valve is incompetent, and when the heart is very feeble, the characters of the pulse, which I have just described, may become variously modified.

5. Capillary and venous pulse.—In aortic regurgitation a pulse wave can sometimes be seen in small vessels, such, for example, as the retinal vessels and the capillaries of the skin, in which, under normal circumstances, pulsation is invisible. The capillary pulsation in the skin is best elicited by drawing the finger over the skin (the skin of the forehead, for example), and producing a capillary blush, which is seen to expand and retract, *i.e.* to pulsate, with each ventricular systole. The capillary pulse is doubtless due to the fact that the large blood wave which is propelled with abnormal suddenness and force, by the dilated and hypertrophied left ventricle into the relaxed arteries, makes its way *as a pulse or wave* into vessels into which, under normal circumstances, the pulse wave does not penetrate.

A venous pulse, synchronous with the systole of the left ventricle, is occasionally seen in the peripheral veins (the veins on the back of the hand for example); it owes its origin to the same cause, *viz.*, the pulse wave passing on right through the capillaries into the venous circulation.

The capillary and venous pulses, which depend on the force with which the blood is propelled into the relaxed arteries, disappear when dilatation becomes excessive, *i.e.* on the failure of compensation.

Should the mitral valve give way, the usual primary and secondary physical signs indicative of that condition will of course be present; the pulse then loses some of these characteristics, though it usually still presents the suddenness of rise and rapidness of fall which it has in the earlier stages of the case.

6. Alterations in the character of the cardiographic tracing.—The character of the cardiographic tracing depends upon (*a*) the condition of the left ventricle (whether hypertrophy or dilatation is in excess), and (*b*) the extent of the incompetence. The most characteristic alteration is the short duration of the diastolic portion of the tracing and the rapid filling of the ventricle during its diastole—alterations which are still more marked when, in addition to the aortic incompetence, mitral regurgitation is present.

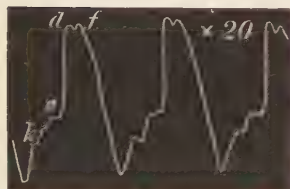


FIG. 214.

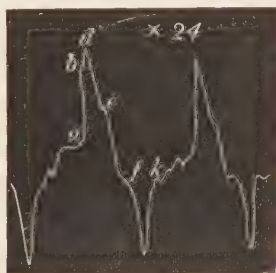


FIG. 215.

FIG. 214.—*Cardiogram from a case of aortic regurgitation.*—(After Galabin.)

‘Thomas S., æt. 45. The diastolic murmur was very loud and accompanied by a thrill felt at the apex, P. 74.’—(*Guy’s Hospital Reports*, 1875, p. 313.)

FIG. 215.—*Cardiogram in a case of aortic regurgitation.*—(After Galabin.)

‘The heart was much dilated, the apex beat being in the sixth intercostal space, and external to the line of the nipple. The pulse tracing showed extreme collapse in the diastolic portion, and an almost entire absence of the tidal wave. From this it may be inferred that the regurgitation was very free, and the contractions of the heart short and incomplete. The tracing is partly inverted, and a retraction occurs during the latter part of systole, followed by a sudden recoil.’—(*Guy’s Hospital Reports*, 1875, p. 312.)

When hypertrophy is in excess of dilatation the summit of the systolic portion of the tracing may be broad ; when dilatation has gained the upper hand, the summit of the systolic portion of the tracing is pointed, and the subsequent descent of the lever rapid. (See figs. 214 and 215.)

In some cases, as in figure 215, the presence of the murmur or thrill is demonstrated in the diastolic portion of the tracing by a series of indentations or serrated curves.

Diagnosis.—When a case of supposed aortic regurgitation comes under notice we have to determine :—

- (1) Is aortic regurgitation actually present ?
- (2) If aortic regurgitation is present, is it due to disease originating in the endocardium, or to disease originating in the aorta ?
- (3) What is the extent and gravity of the lesion ?

Step No. 1.—*Is aortic regurgitation actually present ?*—The solution of this question is seldom attended with any difficulty. The characteristic physical sign of aortic incompetence is, as we have previously seen, a diastolic aortic murmur ; and since a diastolic murmur may (theoretically) be generated at the mitral, tricuspid, and pulmonary orifices, in other words, as the result of mitral stenosis, tricuspid stenosis, and pulmonary regurgitation, an inexperienced observer might suppose that the condition would be likely to give rise to difficulty. As a matter of fact, mitral stenosis does occasionally produce a diastolic murmur, but this murmur has its point of maximum differential intensity in the mitral area, and can be readily distinguished from a diastolic murmur indicative of aortic regurgitation by attention to the other points which have been previously detailed in speaking of the differential diagnosis of mitral stenosis.

Tricuspid stenosis is comparatively rare. So far as I am aware, a diastolic (as distinguished from a presystolic) murmur has not been observed in that condition. But even if a diastolic murmur is sometimes present in tricuspid stenosis, it would be impossible for an experienced observer to mistake

that condition for aortic regurgitation. (This point will be more apparent after the tricuspid lesions have been described.)

A diastolic pulmonary murmur is practically unknown,—and even if it did occur the secondary alterations in the heart and circulation are so totally different from those which are produced by aortic regurgitation that the two conditions could not be confounded.

A more practical question is the differential diagnosis of the double murmur of aortic disease and the double murmur due to pericarditis; but since this point has been previously considered in detail (see p. 328), I need not recapitulate the points of distinction between the two conditions.

Step No. 2.—If aortic regurgitation is present, is it due to disease originating in the endocardium (i.e. to endocarditis), or to disease originating in the aorta (i.e. to atheroma, aneurism, or simple dilatation of the aorta, etc.)?

In attempting to decide this question, we must take into consideration the following points:—

(1) The age, sex, and occupation of the patient, and the previous history of the case.

Youth, the female sex, the fact that the patient has not been subjected to strain, a history of acute rheumatism or of some other affection in the course of which endocarditis is like to arise, are strongly in favour of the endocardial origin of the disease. *Vice versâ* when the patient is a male, when he has been exposed to strain, has suffered from syphilis, or indulged in alcoholic excess, when there is no history of rheumatism or other disease likely to be attended with endocarditis, and more particularly when he is advanced in years, the aortic or arterial origin of the lesion is probable.

(2) The condition of the aortic and peripheral vessels as determined by physical examination, and by the character of the symptoms which are present.

The physical signs of a dilated aorta (see page 696), an atheromatous condition of the superficial vessels, and the presence of symptoms indicative of intra-thoracic pressure, are strongly in favour of the aortic origin of the lesion.

Pain in the region of the heart, and attacks of angina pectoris (which often depend upon disease of the coronary arteries, and therefore upon atheroma) are also in favour of the aortic origin of the lesion.

Step No. 3.—What is the extent and gravity of the lesion?

This question will be more appropriately considered under the prognosis, to which I now pass.

The Prognosis.—The risk of sudden death is very much greater in aortic regurgitation than in any other form of valvular lesion. Barring this risk (which may be considered as accidental, for it is impossible to exclude it even in cases in which the lesion is comparatively slight, and it of course does not always occur even in severe cases), the average prospect of life duration is better in aortic regurgitation than the average prospect in mitral lesions; and life, so long as it lasts, is infinitely more enjoyable.

Individual cases must, however, be judged on their own merits; and in trying to estimate the gravity of the lesion in any individual case which comes before us, we must take into consideration the following points:—

(1) *The extent of the lesion.*—This is estimated by (a) the extent and character of the secondary changes in the left ventricle, (b) the degree of anæmia in the arterial system during the ventricular diastole, and (c) the urgency of the symptoms which are present. The greater the hypertrophy and dilatation of the left ventricle, the greater the arterial anæmia, and the more urgent the symptoms produced by the anæmia, such, for example, as attacks of syncope—the greater the incompetence. A diastolic murmur in the peripheral vessels, a capillary or venous pulse, all indicate extensive regurgitation.¹

(2) *The ætiology of the case.*—The extent of the lesion being equal, aortic incompetence due to aortic or arterial change is a more serious condition than aortic incompetence

¹ I presume, of course, that the hypertrophy and other cardiac changes are due to the aortic regurgitation, and not to other conditions, such as atheroma, for example.

due to endocarditis. When the regurgitation is due to atheroma, the patient is older, and the capabilities of compensation are not so great; the coronary arteries are frequently involved in the atheromatous process, and the nutrition of the heart is therefore interfered with; the risk of arterial rupture (cerebral apoplexy, for example) is very considerable. All of these circumstances make atheromatous aortic regurgitation (the extent of the lesion being the same) more serious than endocarditic aortic regurgitation. A dilated condition of the aortic arch, and especially an aneurismal, as distinct from a uniform dilatation, adds seriously to the dangers of the case.

Regurgitation due to rupture of an aortic segment, is the most serious form of the disease.

(3) *The capabilities of compensation.*—The same general principles which have been laid down in treating of the prognosis of mitral lesions apply here. It must however be remembered, that in aortic lesions the pulmonary, digestive, excretory, and nervous organs, and the walls of the heart itself, are protected from the baneful effects of venous engorgement; so long, therefore, as the mitral valve remains competent, the capabilities of compensation are much greater in aortic than in mitral cases.

(4) *The risk of accidents and complications.*—Barring sudden death and accidental complications due to atheroma, the risk of complications is much less in aortic than in mitral cases.

Treatment.—In cases of aortic regurgitation due to rheumatic valvulitis, little or no drug treatment is required so long as compensation remains good. At this stage of the case, the general rules, which have been recommended for the treatment of organic mitral regurgitation previous to the failure of compensation (see page 467) must be carried out; the main object being to maintain the tissues as a whole, and the cardiac muscle in particular, in the highest possible state of health. Much more exercise can be indulged in and allowed than in cases of mitral disease, in fact, patients affected with this form of aortic incompetence (*i.e.* aortic incompetence the result of valvulitis and before the failure of compensation)

should be encouraged to lead active out-door lives ; and they may engage in any exercise which does not involve strain and which does not over-tax the heart. It is as well to err on the side of caution, for unlike the subjects of mitral incompetence patients affected with aortic incompetence have little or no shortness of breath, and experience no sensations which indicate the limit to which exertion may be safely carried. Violent or prolonged exertion should be avoided.

The tendency to sudden death from syncope and over-distention of the left ventricle must be remembered; and patients affected with this, and indeed with all forms of aortic incompetence, must be emphatically told to avoid everything likely to induce syncope. Excesses of all kinds, but more especially sexual excesses and over-indulgence in tobacco, are to be prohibited ; strong purgatives, Turkish baths or prolonged immersion in the ordinary warm bath, all of which tend to produce fainting, should be avoided.¹

In the cases in which the aortic incompetence is associated with atheroma, the patient should lead a quiet life; in cases of this description, only so much exercise as is required to maintain the general health should be permitted ; everything, such as straining at stool, sudden effort, etc., which suddenly increases the arterial blood pressure, and which is therefore apt to produce rupture of the degenerated vessels, is to be avoided.

In those cases in which the aortic incompetence is associated with aortic aneurism, iodide of potassium and the other measures, which will be detailed when the treatment of aortic aneurism comes under consideration (see page 740), should be adopted. (Iodide of potassium is often very useful in those cases of aortic incompetence which result from increased arterial tension, and in which there is no aneurism.)

When the left ventricle begins to fail, and dilatation to out-strip hypertrophy, cardiac tonics and stimulants are called for.

¹ I have known death result from the exhaustion following coitus, in a patient affected with aortic regurgitation. In another case the patient tumbled down in a faint after a copious watery evacuation of the bowels ; such an accident may of course occur to a healthy person, but is much more likely to arise, and is dangerous, in a person affected with aortic incompetence. In the case to which I refer, the syncope was recovered from,

Arsenic is the remedy which has seemed to me most useful in the earlier stages of this affection; iron is also in many cases beneficial. When arsenic and iron fail, digitalis or some of the other cardiac tonics should be given. Marked improvement generally follows the administration of digitalis, but the remedy should be cautiously and judiciously administered, for large doses are not so well borne in cases of aortic incompetence as in mitral disease. This drug should not be indiscriminately prescribed in aortic regurgitation; and when its administration is necessitated, the dose should be small and the remedy should be discontinued as soon as the required tonic effect has been produced. If these points be attended to, digitalis will often be found pre-eminently beneficial. Alcoholic stimulants are often useful, but, like digitalis, they must be judiciously administered, more especially in those cases in which the aortic incompetence is associated with general atheroma.

For the relief of cardiac pain, which is of frequent occurrence in cases of aortic incompetence, more especially in atheromatous cases, arsenic is most useful; the application of a belladonna plaster to the præcordial region often seems to give relief. When attacks of genuine angina pectoris occur, the measures which will be afterwards recommended for the treatment of that condition must be adopted. (See page 686.)

AORTIC STENOSIS.

Definition.—Narrowing of the aortic orifice.

Ætiology and Pathology.—Narrowing of the aortic orifice may result from acute endocarditis with the formation of luxuriant vegetations, or from chronic endocarditic or atheromatous changes in the valve. The stenosis is, in the majority of cases, combined with incompetence, though exceptions to this, the general rule, are occasionally met with, generally in old people. In some of the combined cases (*i.e.* cases of aortic stenosis and aortic incompetence), stenosis is in excess, but in the majority the regurgitant lesion is the more important. I need not farther consider the latter group of cases, in which

the condition practically corresponds in most cases to (pure) aortic incompetence,¹ but will limit my remarks to those cases in which the stenosis is the only or the predominant lesion, and in which the lesion is a chronic one.² The narrowing may be due to adhesion of the valve segments, as the result of inflammatory changes either during foetal life or after birth, and sclerotic and atheromatous changes in the valve segments and basal ring of the aorta. In many cases these two processes (adhesion of the segments and atheromatous changes) are combined.

Aortic stenosis is, in its pure and typical form, essentially a disease of later life, and is usually combined with general atheroma and calcification of the arteries. Fothergill supposes that 'when the valvulitis tends toward aortic stenosis, the growth commences at the base of the cusps, and from thence spreads towards the free edges; and, synchronously, there is a growth of connective tissue corpuscles in the arterial conus, so that there is stenosis of the conus along with obstruction due to the stiffened valve. . . . On the other hand, in young subjects, the growth commences in the fibrous structure of the free edge of the cusps, extending from the corpora Arantii to the insertion of the cusps in the aortic wall; so that the free edge is thickened, and then in time contracted, consequently the cusps become insufficient, and there is regurgitation through them on the aortic recoil.'³

Pathological Physiology.—The *first* effect of aortic stenosis is of course to interfere with the passage of the blood from the cavity of the left ventricle into the aorta during the ventricular systole; the *second* is to produce a series of changes in the heart and circulation, which are as follows:—

Effect on the left ventricle.—In consequence of the increased effort which is required to force the blood through the stenosed orifice, the left ventricle becomes hypertrophied, its systole is prolonged, and the frequency of the cardiac contractions is

¹ See page 509.

² The acute lesions of the valves are described on p. 368.

³ *Diseases of the Heart*, p. 159.

diminished. (In those cases in which the stenosis is accompanied by regurgitation, the cavity of the left ventricle also becomes dilated, but in stenosis *per se* there is little or no dilatation.) Both changes (hypertrophy of the left ventricle and prolongation of the ventricular systole) are beneficial and compensatory, inasmuch as they allow an increased quantity of blood to pass through the narrowed orifice at each ventricular systole.

Effect on the left auricle, pulmonary circulation, right heart and systemic venous circulation.—So long as the hypertrophy of the left ventricle is good (and in pure cases of aortic stenosis the compensation usually remains satisfactory for a considerable, and in many cases for a long time), the pulmonary and systemic venous circulations are little if at all interfered with, and there are no evident indications of secondary changes in the left auricle or right heart. After the failure of compensation, and when the cavity of the left ventricle becomes dilated, the pulmonary and venous circulations may become engorged, and secondary (relative) mitral incompetence may occur.

Effects on the arterial circulation.—The amount of blood which is propelled into the aorta at each contraction of the left ventricle is, in cases of advanced stenosis of the aortic orifice, smaller than normal, the pulse is therefore diminished in volume, the diminution being directly proportionate to the degree of the stenosis, *less* the extent of the compensatory changes which are present. In addition to a diminution in volume, the pulse wave presents other modifications, which will be presently described. I must, however, here point out that it is only in advanced cases of aortic stenosis that the supply of blood to the arterial system is so much diminished as to produce important alterations in nutrition and in the functional condition of the peripheral tissues and organs. It must, too, be remembered that the subjects of aortic stenosis are, as a rule, old people, who lead quiet, inactive lives, and in whom all the tissue changes are at a minimum.

Symptoms.—So long as compensation is satisfactory, and

the stenosis not very great, there are few if any symptoms. As the lesion progresses, and the supply of arterial blood to the peripheral organs and tissues becomes seriously interfered with, symptoms, more particularly giddiness or fainting, loss of nerve tone and of muscular power, defective (slow) cerebration, muscular twitchings or even epileptiform convulsions—due to defective supply of arterial blood to the brain—coldness of the hands and feet, and symptoms indicative of general debility and impaired nutrition, may occur. When compensation fails, and more especially when the mitral valve gives way, the usual symptoms indicative of engorgement of the pulmonary and systemic circulations arise. Cheyne-Stokes' respiration sometimes develops and ushers in a fatal termination; in other cases complications, such for example as apoplexy, the result of arterial rupture, may arise; indeed many of the symptoms in typical cases of aortic stenosis (*i.e.* pure stenosis in old people) are due to the diseased condition of the peripheral vessels. In other cases accidental symptoms due to embolic plugging of peripheral vessels occur.

Physical Signs.—The *primary* physical sign which results from aortic stenosis is a systolic murmur in the aortic area, which is propagated upwards over the course of the aorta and into the great vessels of the neck. (See fig. 216.) The murmur is usually loud, somewhat harsh in character, though sometimes musical; in other cases it is soft and faint.

The second sound is usually faint and indistinct; in those cases in which the valve is incompetent as well as contracted, it is of course replaced by a diastolic murmur.

In addition to the systolic murmur, a systolic thrill can often be felt at the base of the heart, or over the ascending thoracic aorta.

In consequence of the hypertrophy of the left ventricle, the apex beat is displaced downwards and to the left; the area of the cardiac dulness over the left ventricle is increased,¹

¹ The increased cardiac dulness and displacement of the apex are not so great as in aortic incompetence.

and a powerful, heaving, but slow and deliberate, impulse can in many cases be felt on palpating the præcordia. The apex beat is usually weak and ill-defined, sometimes quite imperceptible, a fact which Traube explains by supposing that the force of recoil or the power which drives the heart in the direction opposite to the systolic outflow, and which is one of the factors in the cardiac impulse, is impeded in aortic stenosis since the size of the aortic opening is smaller, and the resistance to the outflow greater than under normal conditions.¹

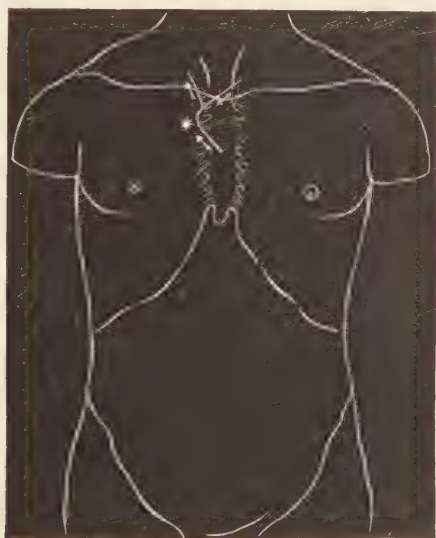


FIG. 216.—Outline figure showing point of differential maximum intensity (*) of the systolic aortic murmur (aortic stenosis); and the directions in which it is propagated.

In typical cases, the pulse is slower than normal; its volume is diminished, but unlike the small pulse of mitral regurgitation it is of good strength (tension), the artery being well filled during the ventricular diastole; the pulse of aortic stenosis is, as a rule, quite regular; its sphygmographic characters are well seen in the following tracings, the most

¹ Quoted by Rosenstein, *Ziemssen's Cyclopaedia*, vol. vi. p. 143.

important alterations being the gradual ascent of the up-stroke corresponding to the deliberate contraction of the left ventricle, the rounded apex, and the long duration of both the ventricular systole and the ventricular diastole. (See figs. 217 and 218.)



FIG. 217.—Pressure $1\frac{1}{2}$ oz.

FIG. 217.—Pulse tracing in aortic stenosis.

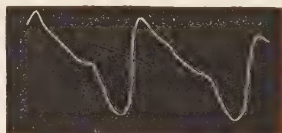


FIG. 218.—Pressure $1\frac{1}{2}$ oz.

FIG. 218.—Pulse tracing in aortic stenosis and dilatation of the arch of the aorta, the latter being the chief lesion.

Diagnosis.—When a systolic murmur is heard at the base of the heart, we have to determine :—

1. Is the murmur generated in the aorta ?
2. If it is generated in the aorta, is it due to organic or functional causes ?
3. If it is due to organic causes, does it depend upon aortic stenosis ?
4. If aortic stenosis is present, what is the extent and gravity of the lesion ?

Step No. 1.—Is the murmur generated in the aorta ?

There is seldom any difficulty in deciding this point, provided that an accurate survey be made of the physical condition of the heart and circulation. Systolic murmurs generated at the mitral, pulmonary, and tricuspid orifices, may, of course, be heard over the aortic valve, but are readily distinguished by attention to their points of maximum differential intensity, their direction of propagation, their sound characters, and the secondary results which they produce in the heart itself and in the peripheral venous and arterial circulations. The chief points of distinction are shown in the following table :—

TABLE VIII.—Differential Diagnosis of Organic Systolic Endocardial Murmurs.

	Point of Differential Maximum intensity.	Direction of Propagation.	Condition of Left Ventricle.	Condition of Right Ventricle.	Left Auricle.	Right Auricle.	Pulmonary Second Sound.	Aortic Second Sound.	Lungs.	Systemic Venous Circulation.	Systemic Arterial System; Pulse.
<i>Aortic Stenosis.</i>	2d right costal cartilage.	Upwards over aorta and into vessels of neck.	Hypertrophied and dilated.	Normal.	Normal.	Normal.	Normal.	Weak, absent or replaced by a murmur.	Normal.	Normal.	Radial pulse, small, of good strength and regular; if lesion severe, anæmia.
<i>Mitral Regurgitation.</i>	Apex.	Upwards and outwards towards left axilla, and to inferior angle of scapula.	Hypertrophied and dilated.	Usually hypertrophied and dilated.	Dilated and hypertrophied.	Sometimes dilated and hypertrophied.	Accentuated.	Usually weak.	Engorged, lung symptoms.	Engorged.	Radial pulse, small, weak, and often irregular. Arterial anæmia.
<i>Pulmonary Stenosis.</i>	3d left costal cartilage.	Upwards and to the left.	Normal, if lesion is primary.	Hypertrophied.	Normal.	Dilated and hypertrophied.	Weak, absent or replaced by a diastolic murmur.	Usually weak.	Anæmic, lung symptoms.	Engorged, if lesion organic.	Radial pulse, small and weak, may be irregular. Arterial anæmia.
<i>Tricuspid Regurgitation.</i>	Lower end of sternum and adjacent cartilages.	Upwards and to the right.	Normal, if lesion is primary.	Hypertrophied and dilated.	Normal.	Dilated and hypertrophied.	Usually accentuated; when regurgitation is primary, weak.	Usually weak.	When lesion secondary, engorged; when primary anæmic; in both cases lung symptoms.	Engorged; pulsation in external jugulars and liver.	Radial pulse, small, weak; may be irregular. Arterial anæmia.

Notes.—This Table only gives the points of distinction in pure, *i.e.* uncomplicated cases.

Step No. 2.—If the murmur is generated in the aorta, is it due to organic or functional causes?

In conditions of anæmia, functional murmurs are sometimes generated at the aortic orifice, and it is very important to distinguish cases of this description from cases in which the murmur is due to organic changes in the valve segments or in the aortic arch.

Now, it is quite possible, that a patient who is affected with aortic stenosis may at the same time be anæmic; and in cases of this description it may be difficult or impossible to arrive at a positive conclusion as to the cause of a systolic murmur heard in the aortic area. There is usually no difficulty in distinguishing uncomplicated cases (pure cases of aortic stenosis on the one hand from pure cases of anæmic basic murmurs, such as are met with in the course of chlorosis, or progressive pernicious anæmia, on the other) by attention to the following points:—

1. *The age and general condition of the patient.*—In chlorosis and progressive pernicious anæmia, which are the forms of general anæmia in which basic murmurs are most apt to arise, the patient is, as a rule, young, or in active adult life, the skin often has a lemon yellow tinge, and there is no wasting of subcutaneous fat. Whereas in the anæmia which is associated with aortic stenosis, the patient is generally old, as a rule spare, the subcutaneous fat in particular being generally scanty, and general atheroma often present.

2. *The condition of the left ventricle, and of the right heart.*—In anæmia the left ventricle is dilated rather than hypertrophied, whereas in aortic stenosis the hypertrophy is generally pure. In anæmia the right heart is also dilated and hypertrophied, whereas in aortic stenosis it is usually quite normal.

3. *The character of the pulse.*—In aortic stenosis the left ventricle contracts with much deliberation, the pulse is slower than normal, and the apex of the sphygmographic tracing is usually rounded; whereas in anæmia the ventricle contracts in a sharp, irritable manner, especially when the patient is

agitated; the frequency of the cardiac contraction is, under such circumstances, considerably increased, and the apex of the sphygmographic tracing is more pointed than normal. Contrast figure 217 with figures 219 and 220.

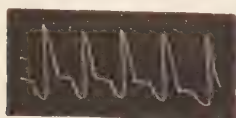


FIG. 219.

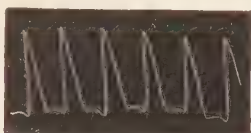


FIG. 220.

FIGS. 219 and 220.—Pulse tracings from a case of progressive pernicious anæmia.

Fig. 220 was taken shortly before death; the artery is very empty during diastole.

4. *The sound character of the murmur, the presence of a systolic basic thrill, and other murmurs over the heart.*—The aortic murmur of anæmia is almost invariably soft and badly propagated, and is often quite inaudible in the carotids, whereas the murmur of organic stenosis is usually loud and harsh, and is well propagated over the course of the aorta and into the great vessels of the neck.

A *systolic thrill* can frequently be felt in aortic stenosis. An aortic murmur due to anæmia is usually (? invariably) accompanied by a venous hum in the neck, by a pulmonary systolic murmur, and very often by mitral and tricuspid systolic murmurs. An aortic systolic murmur due to stenosis is often associated with a diastolic aortic murmur.

5. *The effects of treatment.*—Many cases of anæmia are, as we have previously seen, completely curable; in cases of this description the basic murmur completely disappears under treatment, whereas in aortic stenosis the lesion always remains.

6. *The nature of the symptoms.*—In anæmic cases the least exertion produces shortness of breath and palpitation, and there is often some swelling of the feet. These symptoms are not present in aortic stenosis, although they may, as we have seen, occur in the later stages of the case.

Step No. 3.—The murmur is due to organic causes; does it depend upon aortic stenosis?

The other organic conditions which may produce an aortic systolic murmur, independently of disease of the aortic orifice, are :—

1. *Dilatation of the ascending thoracic aorta.*—Dilatation of the aorta (which is frequently associated with aortic stenosis) usually gives rise to increased dulness over the course of the aorta, increased pulsation in the aortic notch; a systolic thrill, in addition to the murmur, is usually present, and there may be symptoms and signs indicative of the pressure of the dilated vessel upon the surrounding structures and parts. When the aortic segments are healthy, the aortic second sound is loudly accentuated, whereas in aortic stenosis it is usually weakened or effaced. Both conditions may be accompanied by a diastolic aortic murmur, indicative in the former, of relative incompetence, in the latter, of regurgitation due to organic changes in the valve flaps.¹

2. *Constriction of the aorta by the pressure of an intra-thoracic growth.*—Cases of this description could hardly be mistaken for cases of pure stenosis (*i.e.* stenosis without aortic dilatation); there may be considerable difficulty in distinguishing them from aneurismal dilatation of the aortic arch, but this point will be afterwards considered in detail (see page 726).

Prognosis.—Aortic stenosis is the least serious of all the organic valvular lesions of the heart, and its average duration is longer than that of other lesions; it is often unattended by any serious or distressing symptoms, and it has not the same tendency to produce sudden death as aortic regurgitation.

The gravity of each individual case must of course be determined by observing :—

(1) The extent of the lesion. (The amount of the secondary hypertrophy of the left ventricle, and the characters of

¹ The exact nature of the symptoms and physical signs which characterise dilatation of the aorta will be more particularly described when the diseases of the aorta are treated. (See p. 726.)

the pulse, are the points to which attention must be specially directed in order to determine the extent of the lesion.)

(2) The capabilities of compensation and resistance.

(3) The presence of complications. All of these points have been so fully considered in treating of the other valvular lesions, that they need not be again detailed.

Treatment.—The general plan of treatment which has been recommended for aortic regurgitation must be carried out. When the left ventricle begins to fail, digitalis must be freely given; but it must be discontinued as soon as its tonic effect has been produced, the prolonged administration of the drug increases the arterial tension, and theoretically, at all events, may be supposed to increase the risk of rupture of the diseased peripheral vessels.

TRICUSPID INCOMPETENCE.

Definition.—Tricuspid incompetence includes all those conditions which interfere with the perfect closure of the tricuspid valve apparatus, and allow a regurgitant blood current to pass from the cavity of the right ventricle into that of the right auricle during the ventricular systole.

Ætiology and Pathology.—The tricuspid valve apparatus is constructed on the same plan as the mitral valve apparatus,¹ the only differences being (1) that the tricuspid valve has three segments, and (2) that the muscular wall of the right ventricle, or, as we may term it, the tricuspid sphincter, is thinner and weaker than the muscular wall of the left ventricle (or mitral sphincter), and that relative and muscular incompetence are, therefore, more readily produced at the tricuspid than at the mitral orifice; it is indeed probable, that tricuspid incompetence frequently occurs even in perfect health as the result of the temporary over-distention of the right ventricle. King was the first to direct attention to this,—the safety valve action of the tricuspid, as he termed it.

¹ The reader is recommended to refer to the detailed description of the anatomy and physiology of the mitral valve apparatus described on p. 421.

Tricuspid incompetence may, therefore, result from :—

(1) Organic changes in the valve segments.

(2) 'Muscular' and 'relative' incompetence.

Now incompetence due to organic changes in the tricuspid valve segments is rare ; it occasionally occurs as a congenital condition, *i.e.* as the result of inflammatory changes during intra-uterine life ; sometimes also as the result of endocarditis in after life ; and the same causes which produce endocarditis at the mitral orifice¹ are the causes of tricuspid endocarditis. The pathological appearances in both conditions (tricuspid and mitral endocarditis) are the same. I must further repeat that I am disposed to think that acute inflammation of the tricuspid valve is much more common than is generally supposed ; and that, like the endocarditis of chorea, it usually subsides without leaving any permanent structural changes.

'Muscular' and 'relative' incompetence of the tricuspid are, on the contrary, more common than 'muscular' and 'relative' incompetence of the mitral, and may result from :—

(a) Structural changes in the muscular wall of the right heart, amongst which fatty changes, the result of anæmia, are perhaps the most important. In these cases we may term the condition *primary* 'muscular' and 'relative' incompetence.

(b) Dilatation of the cavity of the right ventricle, the result of an obstruction in front, *i.e.* in the lungs or at the mitral orifice. In these cases, in which we may term the condition *secondary* 'muscular' and 'relative' incompetence, there are usually also degenerative changes in the cardiac muscle, in consequence of which the right ventricle becomes dilated rather than hypertrophied. I must repeat, that this form of tricuspid regurgitation, which depends upon dilatation and degeneration of the right ventricle, and which is secondary to an obstruction either in the lungs or at the mitral orifice, is extremely common ; and since the primary causes of the condition (*i.e.* pulmonary or mitral lesions) are in some cases completely curable, the tricuspid incompetence may also be a temporary and curable condition.

¹ See p. 425.

Pathological physiology.—The *first* effect of tricuspid regurgitation is to allow a regurgitant blood current to pass from the cavity of the right ventricle to that of the right auricle during the ventricular systole. The *second* is to produce a series of changes in the heart and circulation, which are as follows:—

Effect on the heart.—The right auricle becomes dilated, and in some cases also hypertrophied. The hypertrophy is, however, as a rule, slight, in consequence of the facts, *firstly*, that the wall of the auricle is extremely thin and incapable of great hypertrophy; and *secondly*, and more particularly, that in cases of tricuspid incompetence the conditions for the production of satisfactory hypertrophy (a healthy condition of the system as a whole and of the heart in particular) are seldom present. In many cases the muscle fibre is degenerated, and the nutrition of the heart and the general powers of compensation and resistance are weakened by the primary disease (such for example, as long continued venous congestion, the result of a chronic mitral lesion), on the top of which, so to speak, the tricuspid regurgitation is added.

Theoretically, the right ventricle ought to become hypertrophied and dilated as the result of tricuspid incompetence, just as the cavity of the left ventricle becomes dilated and hypertrophied as the result of mitral incompetence; as a matter of fact, however, hypertrophy does not as a rule occur, *firstly*, because the powers of compensation are feeble; *secondly*, because, in secondary cases at all events, the right ventricle has already put forth all its efforts, and has become as fully hypertrophied as it can become (in order to overcome the obstruction in front) before the tricuspid incompetence is established.

Effect on the circulation in front.—As soon as tricuspid incompetence is established, the quantity of blood propelled into the pulmonary circuit becomes diminished; the left heart consequently receives less blood, and the supply of blood to the systemic arterial circulation is lessened.

Effect on the venous circulation.—The effect which tricuspid incompetence produces upon the systemic venous circulation

is extremely serious. At each contraction of the right ventricle a back-wash (the amount of the regurgitant current being directly proportionate to the extent of the tricuspid leak and the power of the right ventricle) passes through the cavity of the right auricle into the superior and inferior venæ cavæ ; in fact, with the occurrence of tricuspid regurgitation the last cardiac barrier, which protects the systemic venous circulation from the injurious effects of backward pressure, is removed, and all the secondary changes in the peripheral tissues and organs, which I have detailed under the head of mitral incompetence, become seriously aggravated. The engorgement of the veins is now, it must be remembered, no longer passive, but at each systole of the heart a regurgitant current is forced right into the venous trunks, and the peripheral tissues and organs are then only protected from the direct effects of the 'back-wash' by the valves of the veins themselves.

Clinical History.—Tricuspid incompetence is (as I have so repeatedly stated), with rare exceptions, a secondary condition. The symptoms therefore which it produces are, in the great majority of cases, superadded to the symptoms of the primary affection to which the tricuspid incompetence is secondary. The exact nature of the primary symptoms varies, of course, with the exact nature of the primary lesion, but, for our present purpose, we may suppose that the primary condition is a progressive organic lesion of the mitral orifice (mitral stenosis or mitral incompetence). With the occurrence, then, of tricuspid incompetence, the dropsy and effusion into the serous cavities become greater, the dyspnœa and other respiratory troubles are increased, and hæmoptysis due to pulmonary apoplexy is apt to arise ; the symptoms due to venous engorgement of the digestive and urinary organs and of the nerve centres become aggravated ; in short all the more serious symptoms, which I have previously detailed as characteristic of the terminal periods of a progressive mitral lesion, are fully developed. In cases of this description (progressive mitral disease to which tricuspid regurgitation is super-added) a fatal termination is soon reached.

When the tricuspid incompetence depends upon temporary and curable conditions, the severity of the symptoms is not so great; in conditions of anæmia, for example, a slight degree of incompetence at the tricuspid orifice is of frequent occurrence, and is not necessarily attended with serious symptoms. It must, too, be remembered, that temporary tricuspid regurgitation may occur in the course of organic lesions of the mitral orifice, as the result, for example, of an attack of acute bronchitis or any other complication which produces a temporary failure of compensation and throws a sudden strain on the right heart. In estimating the prognosis of tricuspid incompetence, each individual case must be judged on its own merits, and the exact causation of the tricuspid lesion taken into account.

Physical signs.—The *primary* physical signs indicative of tricuspid regurgitation are:—(1) a systolic murmur in the tricuspid area, and (2) true venous pulsation in the neck.

The murmur is soft and blowing; its point of maximum differential intensity is the lower end of the sternum or adjacent costal cartilages; and its direction of propagation upwards and to the right. (See fig. 221.)

The *venous* pulsation is usually best marked in the external jugular vein on the right side, but is frequently also seen in the corresponding vein on the left side and in the internal jugulars. The pulsation is synchronous with the contraction of the right ventricle, and usually, but not invariably, persists after the lower part of the vein has been emptied by the pressure of the finger, the return current of blood being arrested by pressure on the vein in the manner previously described. Dr Hilton Fagge points out, that in those cases in which the right ventricle is acting feebly or in which the tricuspid leak is slight, the back wave in the veins may not be sufficiently strong to distend the whole of the emptied vein, and so pulsation in the neck may not be observed. Again, in the slighter degrees of tricuspid incompetence, the valves at the junction of the external jugular and subclavian veins may still be competent; and in cases of this description

simple distention or pulsation in the venous bulb, is only observed.

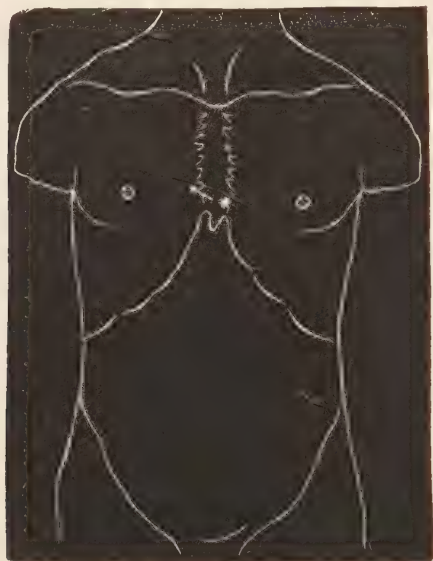


FIG. 221.—Outline figure showing point of differential maximum intensity (*) of the systolic tricuspid murmur.

In addition to the venous pulsation in the neck, true venous pulsation in the liver, causing an expansile pulsation of the organ synchronous with the contractions of the right ventricle, is in many cases also present.

Besides the systolic tricuspid murmur and venous pulsation, increased dulness over the lower end of the sternum and adjacent costal cartilages (due to enlargement of the right heart) is also observed;¹ and the impulse of the right heart can often be seen and felt in the pit of the epigastrium. The radial pulse is small, weak, and very often irregular, in fact, it usually presents the characters which are present in advanced stages of mitral regurgitation.

¹ When the lungs are emphysematous, increased dulness over the præcordia may not be present.

Diagnosis.—The steps in the diagnosis of tricuspid regurgitation are :—

- (1) Is tricuspid incompetence actually present?
- (2) If tricuspid incompetence is present, is the condition primary (*i.e.* due to changes originating in the right heart) or secondary?
- (3) If secondary, what is the nature of the primary condition?
- (4) What is the extent and gravity of the lesion?

Step No. 1.—*Is tricuspid incompetence actually present?*—When a systolic tricuspid murmur and true venous pulsation in the neck or in the liver are present, this question is at once decided in the affirmative, provided, of course, that the observer is satisfied that the systolic murmur is actually a tricuspid murmur;¹ and that the venous pulsation is not one of the false forms of venous pulsation which have been previously described.²

Step No. 2.—*If tricuspid regurgitation is present, is the condition primary (i.e. due to changes originating in the right heart) or secondary to some other condition?*

Step No. 3.—*If secondary, what is the exact nature of the primary lesion?*

In attempting to solve these questions it must be remembered :—(1) that primary tricuspid regurgitation (*i.e.* tricuspid incompetence due to changes originating in the right heart) is comparatively rare; whereas the secondary form of the lesion is common; and (2) that the primary form of the disease is occasionally (more frequently I believe than is generally allowed) met with as an acute affection in the course of rheumatic fever and as a chronic lesion in anæmia and other conditions in which the muscular fibre of the right heart is

¹ The differential diagnosis of pericardial murmurs and endocardial (*e.g.* tricuspid) murmurs is set forth on page 331; while the points of distinction between the different forms of systolic endocardial murmurs are detailed on page 527.

² See page 291.

degenerated. It is also sometimes associated with congenital stenosis of the pulmonary artery.¹

The circumstances under which the tricuspid regurgitation is established, the condition of the pulmonary second sound, and more especially the presence or absence of any disease in the lungs or at the mitral orifice, likely to produce secondary changes in the right heart, are the points to which attention must be directed in order to determine whether the tricuspid lesion is primary or secondary.

In some cases in which cyanosis, dropsy, and tricuspid regurgitation are present, it may be difficult to determine whether the primary lesion is a congenital cardiac affection, such as stenosis of the pulmonary artery with a patent condition of the foramen ovale; or a primary affection of the lungs, such as chronic bronchitis and emphysema; or an advanced mitral stenosis. The points to which attention is to be directed in order to solve this question are given on page 555.

Step No. 4.—What is the extent and gravity of the lesion?
—This question will be more appropriately considered under the prognosis, to which I now pass.

Prognosis.—In considering the prognosis of tricuspid incompetence it is essential to remember (1) that in some cases the regurgitation is due to temporary and curable conditions, such for example as a chlorotic condition of the right heart; and (2) in other cases it is secondary to progressive and incurable organic disease, such for example as mitral stenosis.

In order to give a satisfactory opinion, therefore, as to the significance of tricuspid regurgitation in any individual case, it is essential, in the first place, to determine the exact cause of the condition, and whether that cause is removable (*i.e.* curable) or not. In deciding this question attention must be directed to the points detailed in speaking of the diagnosis

¹ In many of these cases the dilatation of the right heart, on which the tricuspid incompetence partly depends, is due to a double cause, *viz.*, degeneration of the wall of the right ventricle and obstruction in front (in the lungs or at the mitral orifice); in short, in many cases of this description, the tricuspid incompetence is partly primary and partly secondary.

of tricuspid incompetence and the diagnosis and prognosis of 'relative' and 'muscular' incompetence of the mitral orifice.

If we come to the conclusion that the tricuspid regurgitation is due to incurable conditions (if, for example, it occurs in the course of advanced progressive mitral disease), the prognosis is most unfavourable ; indeed tricuspid incompetence is, under such circumstances, of the gravest possible significance, for, with the removal of the tricuspid barrier, the full force, so to speak, of the backward pressure falls directly upon the venous system, and all the grave symptoms which result from venous engorgement become seriously aggravated.

Treatment.—Tricuspid incompetence is to be treated in the same way as mitral incompetence. (See page 469.)

TRICUSPID STENOSIS.

Definition.—Narrowing of the tricuspid orifice.

Ætiology and Pathology.—Stenosis of the tricuspid orifice is sometimes, though very rarely, present at the time of birth. In cases of tricuspid stenosis, in which the condition results from foetal endocarditis, the mitral valve is generally also constricted ; in other cases, pulmonary stenosis and a patent condition of the foramen ovale are present.

In the majority of cases of tricuspid stenosis, the condition probably results from endocarditis after birth. Slight degrees of narrowing are probably by no means so rare as was at one time supposed. Dr Bedford Fenwick, for example, has stated that forty-six cases of tricuspid stenosis have been recorded since the year 1825, and from an analysis of these cases he concludes that the greater number of them, at all events, were due to changes after birth ; in all of the forty-six cases, the mitral valve was similarly affected, and in nearly all, the degree of constriction was greater at the mitral than at the tricuspid orifice.¹

Professor Gairdner has recorded a case (the first case I believe in which the diagnosis of tricuspid stenosis was made

¹ *Lancet*, January 22, 1881, p. 137.

during life) in which the narrowing was due to the pressure of a tumour on the valve segments.

The acquired (non-congenital) form of tricuspid stenosis is like mitral stenosis, much more common in females than in males. The causes of the two conditions (*i.e.* of mitral stenosis and tricuspid stenosis) and the structural changes in the valve segments, are the same; the extreme narrowing, however, which is so common at the mitral, is extremely rare at the tricuspid orifice, a fact which is probably to be explained by the lesser amount of strain to which the segments of the tricuspid valve are subjected.

Pathological physiology.—The effect of tricuspid stenosis is, of course, to interfere with the passage of the blood from the right auricle to the right ventricle, and so through the lungs to the left heart. When the stenosis is congenital, and associated as it sometimes is with pulmonary stenosis, the foramen ovale remains open, and the blood makes a short cut through this channel to the left heart.

When the condition is due to changes after birth, the left auricle is almost always very much dilated. The free supply of blood to the lungs is interfered with in proportion to the degree of the stenosis, and engorgement of the systemic venous circulation is, of course, present. Probably in all cases (in all cases, at all events, of the acquired form), the tricuspid valve is also incompetent.

Symptoms.—In the congenital cases, the symptoms are practically identical with those of pulmonary stenosis, which I will presently describe. In the acquired form, the symptoms are usually those of mitral disease *plus* those of tricuspid incompetence.

Physical Signs.—In one or two cases which have been reported, a presystolic murmur in the tricuspid area has been present, and the condition has been diagnosed during life; in the great majority of cases the condition has not been recognised during life, or has been diagnosed as tricuspid

regurgitation—the stenosis is seldom sufficiently great to cause a presystolic murmur, but a systolic murmur due to associated incompetence is very frequently present.

Diagnosis.—The condition can only be recognised with certainty when a presystolic murmur distinctly localised to the tricuspid area is present. The condition may be suspected when signs of great dilatation of the right auricle and engorgement of the systemic venous circulation are present; and when the tricuspid incompetence, which is almost invariably present in cases of tricuspid stenosis, does not seem sufficient to account for these conditions (in a case of tricuspid incompetence, for instance, in which the right auricle and venous circulations are much engorged, but in which the venous pulse in the neck is feebly marked). If, again, there is reason to believe that the tricuspid incompetence is due to organic changes in the valve segments, rather than to relative or muscular incompetence, the presence of some degree of stenosis as well as incompetence may be legitimately suspected.

Prognosis.—The prognosis is unfavourable; the majority of cases terminate fatally soon after the symptoms of systemic venous engorgement have become prominent.

Treatment.—Little can be done in the way of treatment; the same means which have been recommended for the treatment of advanced mitral disease, and of tricuspid incompetence, are to be adopted.

PULMONARY STENOSIS.

Ætiology.—Stenosis of the pulmonary artery is occasionally, though very rarely, produced after birth, but is more frequent as the result of disease or arrested development during intra-uterine life. Indeed stenosis or complete occlusion (atresia) of the pulmonary orifice or pulmonary artery, with certain secondary changes, such as a patent foramen ovale, or an imperfect intra-ventricular septum, is the condition which is present in the great majority of cases of congenital heart disease.

Acquired (non-congenital) stenosis is occasionally produced in the course of acute ulcerative endocarditis, the orifice being obstructed by luxuriant vegetations; it is sometimes also caused by chronic inflammatory and atheromatous changes at the pulmonary orifice, identical in character and results with the chronic inflammatory and atheromatous changes which are so very much more frequent at the orifice of the aorta. I must repeat, however, that pulmonary stenosis due to any of these conditions is extremely rare.

Congenital stenosis usually results from foetal endocarditis or myocarditis; but it may probably also be due, as Peacock theorised, to defective development of the bronchial arch from which the ductus arteriosus is formed; in such a condition the pulmonary artery would of course receive a much smaller supply of blood than usual, and would therefore be imperfectly developed.¹

Pathology.—In some cases it is extremely difficult to say from the mere pathological characters of the lesion, whether the stenosis belongs to the congenital or to the acquired forms of the disease. When the lesion is limited to the valve segments and basal ring, the condition is probably acquired, for foetal endocarditis is rarely so limited, and the congenital form of the disease is generally, if not always, attended by other changes, such as alterations in the trunk of the pulmonary artery, the sinus arteriosus of the ventricle, and the persistence of some of the foetal openings in the heart. Kussmaul, quoted by Lebert, sums up the points of distinction between the two forms in the following propositions: 'This

¹ During intra-uterine life, the right heart is, as I have previously explained, much more apt to be affected by inflammation (endocarditis and myocarditis) than the left; the greater liability being due to the fact that before birth the valves on the right side are subjected to more strain than those on the left, the right heart being more active than the left. Peacock further supposes that in consequence of the temporary alterations of the blood pressure which are apt to occur in the umbilical arteries and placenta, disease at the base of the pulmonary artery (which is directly continuous with the descending aorta and umbilical arteries) may be established, just as disease of the base of the aorta and aortic valves may result in after life from increased blood pressure (sudden strain, etc.), within the systemic arterial system.

affection of the heart is the more surely congenital :—1st, when the birth was near the normal end of pregnancy ; 2d, the sooner after birth cyanosis and other tokens of heart disease, collectively called physical symptoms of stenosis of the pulmonary artery, are perceived ; 3d, when the foramen ovale and the ductus arteriosus Botalli are both open, or, indeed, only the latter ; 4th, when the opening of the foramen ovale is proportionately large, the ductus being closed, and especially when its size depends on want of the fleshy substance of the septum ; 5th, when the valves of the pulmonary artery show anomalies of structure that are evidently congenital ; 6th, when the trunk of the pulmonary artery is decidedly contracted and its walls are too thin ; 7th, when the right ventricle appears contracted or stunted.¹

The appearances which are met with in congenital stenosis differ very considerably in different cases. The most common condition is that in which the pulmonary artery is distinctly differentiated from the aorta, and narrowed or completely occluded. In cases of this description the valve segments may be fused together, or irregular in development ; the foramen ovale is usually patent, the ductus arteriosus is in some cases open, in others closed ; there is sometimes a deficiency in the intra-ventricular septum.

In other cases, the stenosis is chiefly situated in the right conus arteriosus, which may appear to be a third ventricle cut off from the other two. In cases of this description the intra-ventricular septum is usually deficient, the foramen ovale usually open, the ductus arteriosus sometimes open, sometimes closed. The trunk of the pulmonary artery is usually constricted, and the coats of the vessel thinner than normal.

In a third group, still more striking anomalies are found. In some the division of the common truncus arteriosus into the pulmonary artery and aorta is incomplete ; in others, in addition to the stenosis or complete occlusion of the pulmonary artery, the heart may only consist of two cavities, or, there may be one ventricle and two auricles, or two ventricles and one auricle ; in others again, in addition to the stenosis of the

¹ *Ziemssen's Cyclopadia*, vol. vi. p. 318.

pulmonary artery, the aorta and pulmonary artery are transposed, or have some other abnormal connections with the heart.

The appearances which the pulmonary orifice presents in cases of congenital stenosis are beautifully represented in figs. 222 to 228, which, through the courtesy of his executors, I am able to reproduce from the late Dr Peacock's great work *On Malformation of the Human Heart*.

Pathological physiology.—In the *acquired* form of stenosis the same changes are observed in the right heart and pulmonary artery, which are seen in the left heart and aorta as the result of aortic stenosis; in both cases regurgitation is very generally combined with constriction.

The right ventricle thus becomes hypertrophied and more or less dilated. When the stenosis is considerable, the

Description of figs. 222, 223, 224 and 225, which illustrate Constriction of the Pulmonary Orifice. (After Peacock.)

FIG. 222.—'Diagram to show the extreme constriction at the commencement of the conus arteriosus, the atrophy of that portion of the ventricle, and the form of the arterial opening in Case VIII., p. 84 (in Dr Peacock's work *On Malformation of the Human Heart*). The pulmonary valves are only two in number, and one of them displays appearances of imperfect division. The subject of the disease was a boy 7 years old.

a, Rudimentary conus arteriosus.

b, Piece of wood passed through opening in septum between the conus and the sinus of the right ventricle.'

FIG. 223 —*Heart in Case XIII., described at p. 112, in Dr Peacock's work On Malformation of the Human Heart.*

'The preparation is numbered B 3 in the Museum of the Victoria Park Hospital. The septum of the ventricles is entire, yet the hypertrophy of the right ventricle is seen to be very great. The young man who was the subject of the disease died at the age of 20.

a, The right ventricle laid open to show the great hypertrophy of its parietes.

b, The pulmonic orifice.'

FIG. 224.—*The orifice of the pulmonary artery (Case XIII.) as seen from above.*

'The union of the three valves into one, the frena or bands which mark the imperfect division of the segments, the thickening of the whole of the valves, and the form of the orifice, are well shown in this drawing. The aperture is seen also to have been permanently patent.'

FIG. 225.—'The foramen ovale in the same case, showing that the process of closure has never been completed, the cornua of the valve, *a*, *a*, still remaining widely apart.'



FIG. 222.

FIG. 223.

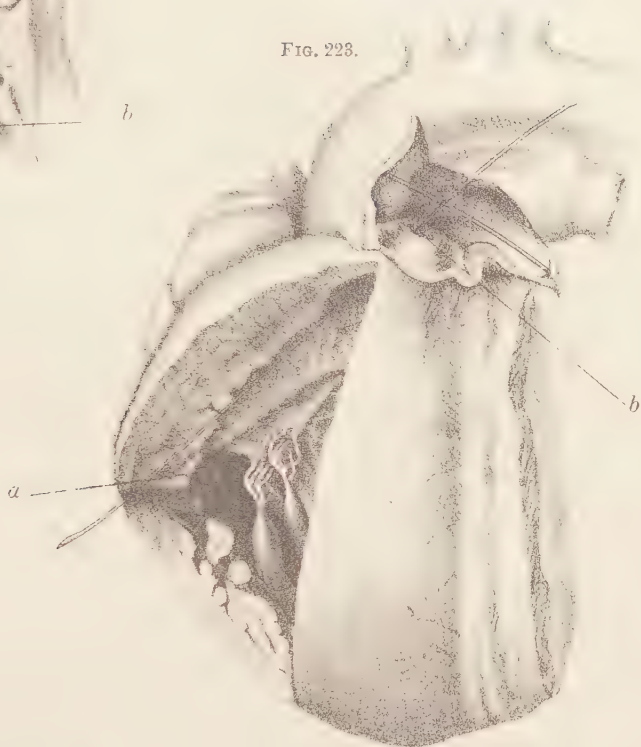


FIG. 225

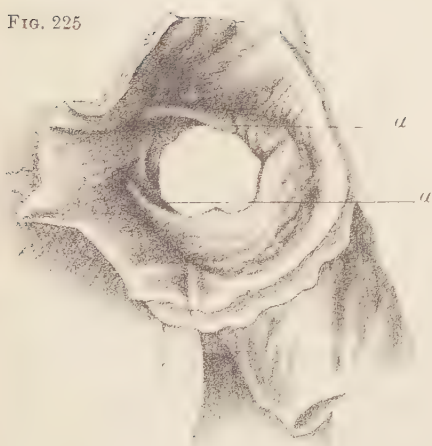
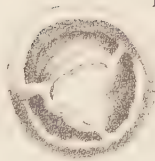


FIG. 224.



FIGS. 222, 223, 224, and 225, illustrate *Stenosis of the Pulmonary Orifice*. (After Peacock.)
For description see page 544.



FIG. 226.—*Deficiency in the septum ventriculorum from a specimen exhibited at the Pathological Society, by Dr Quain, in 1856.*

‘The preparation was taken from a youth eighteen years of age, who died of phthisis at the Brompton hospital, under the care of Dr Cursham. He had been cyanotic since he was two years of age. The ‘aperture was sufficiently large to permit a florin to pass through,’ and was remarkable for occupying the whole of the undefended space and for its regular triangular form.’—(*On Malformation of the Human Heart*, by Dr Peacock, p. 31.)

FIG. 227.

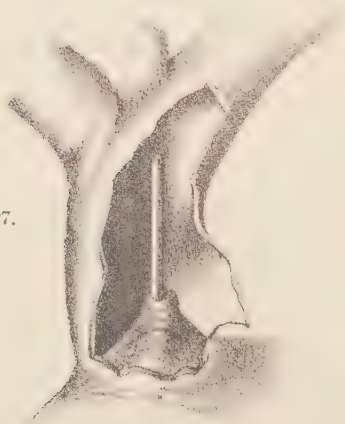


FIG. 227.—*Constriction of the pulmonary orifice. (After Peacock.)*

‘The pulmonary valves are united so as to form an infundibular or funnel-shape opening from the ventricle into the artery; the ductus arteriosus is pervious, and the foramen ovale open.’

FIG. 228.



FIG. 228.—For description see page 545.

pulmonary circulation is imperfectly supplied with blood; the tricuspid may become incompetent, and the systemic venous system engorged. Compensation in cases of this description is chiefly effected by the hypertrophy of the right ventricle.

In *congenital* cases, the secondary results, and the manner in which compensation is established, vary in different cases, and depend chiefly upon the period of intra-uterine life at which the stenosis of the pulmonary artery is established.

But in order that these points may be thoroughly understood, it is necessary to remember the course of the circulation during intra-uterine life. It is represented in fig. 229, and is as follows:—

Course of the Blood in the Fœtus.—The right auricle of the fœtal heart receives blood from the two venæ cavæ and the coronary vein. The blood brought by the superior cava is simply the venous blood returned from the head and upper half of the body; whilst the inferior cava, which is considerably larger than the superior, conveys not only the blood from the lower half of the body, but also that which is returned from the placenta and from the liver. This latter stream of blood reaches the vena cava inferior, partly by a direct passage—the *ductus venosus*—and partly by the hepatic veins, which bring to the vena cava inferior all the blood circulating through the liver, whether derived from the supply of placental blood entering that organ by the umbilical vein, or proceeding from the vena portæ or hepatic artery.

The blood of the superior vena cava, passing from the right auricle into the right ventricle, is thence propelled into the trunk of the pulmonary artery. A small part of it is distributed through the branches of that vessel to the lungs, and returns by the pulmonary veins to the left auricle; but as these vessels remain comparatively undilated up to the

Description of Fig. 228.

Contraction of the outlet of the right ventricle seen from above. (After Peacock.)

The constriction, which was very great, was situated at the base of the pulmonary cusps, and was formed by a muscular band covered by fibrous tissue; the edges of the opening were studded with warty vegetations. Immediately beyond the constriction the passage expanded, so that the valves themselves freely admitted the forefinger between them. The segments were two in number, and one of them displayed some remains of a frenum or band on the upper surface. Except being somewhat thickened and opaque, they were free from disease. There was a deep sinus behind each of them. The pulmonary artery was of small size.—For further description of the case, see Dr Peacock's work *On Malformation of the Human Heart*, p. 55.

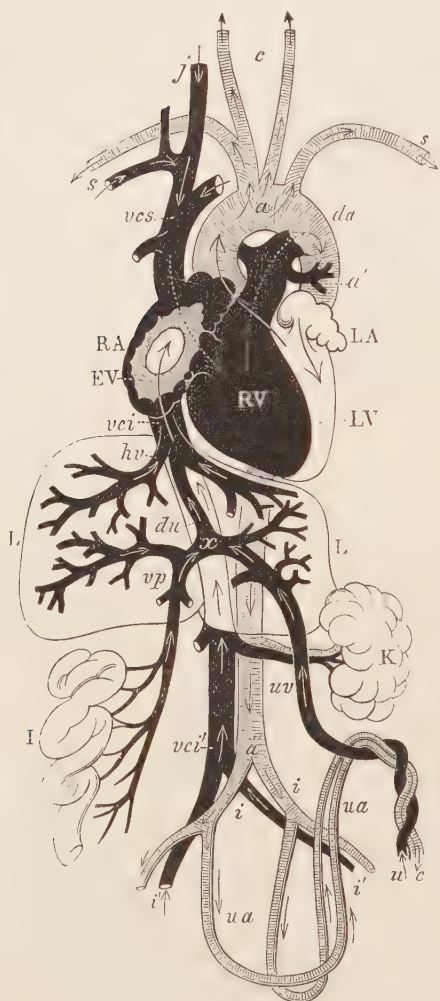


FIG. 229.—Diagrammatic outline of the organs of circulation in the fetus of six months. (After Quain, slightly modified.)

RA, right auricle of the heart; RV, right ventricle; LA, left auricle; EV, eustachian valve; LV, left ventricle; L, liver; K, left kidney; I, portion of small intestine; *a*, arch of the aorta; *a'*, its dorsal part; *a''*, lower end; *vcs*, superior vena cava; *vci*, inferior vena where it joins the right auricle; *vci'*, its lower end; *s*, subclavian vessels; *j*, right jugular vein; *c*, common carotid arteries; four curved arrow lines are carried through the aortic and pulmonary opening, and the auriculo-ventricular orifices; *da*, opposite to the one passing through the pulmonary artery, marks the place of the ductus arteriosus; a similar arrow line is shown passing from the vena cava inferior through the fossa ovalis of the right auricle, and the foramen ovale into the left auricle; *hv*, the hepatic veins; *vp*, vena portæ; *x* to *vci*, the ductus venosus; *uv*, the umbilical vein; *ua*, umbilical arteries; *uc*, umbilical cord cut short; *i* *i'*, iliac vessels.

time of birth, by far the larger part passes through the ductus arteriosus into the dorsal aorta, and is thence distributed in part to the lower half of the body and the viscera, and in part is conveyed along the umbilical arteries to the placenta. From these several organs it is returned by the vena cava inferior, the venæ portæ, and the umbilical vein; and, as already noticed, reaches the right auricle through the trunk of the inferior cava.

Of the blood entering the heart by the inferior vena cava, only a small part is mingled with that of the superior cava, so as to pass into the right ventricle; by far the larger portion, directed by the Eustachian valve through the foramen ovale, flows into the left auricle, and thence, together with the small quantity of blood returned from the lungs by the pulmonary veins, passes into the left ventricle, from whence it is sent into the arch of the aorta, to be distributed almost entirely to the head and upper limbs.

Sabatier was the first to call attention particularly to the action of the Eustachian valve in separating the currents of blood entering the right auricle by the superior and inferior venæ cavæ. This separation, as well as that occurring between the currents passing through the aortic arch and the ductus arteriosus into the descending aorta, was illustrated experimentally by John Reid. A striking confirmation of the extent to which the last mentioned division of the two currents of the foetal blood may take place, without disturbance of the circulation up to the time of birth, is afforded by the examples of malformation in which a complete obliteration has existed in the aortic trunk immediately before the place of the union of the ductus arteriosus with the posterior part of the aortic arch.¹

Now when the stenosis of the pulmonary artery is produced before the end of the third month of intra-uterine life, *i.e.* before the separation of the two ventricles is completed, the intra-ventricular septum remains imperfect, the aperture between the two ventricles being a round hole with smooth edges. In cases of this description, the blood, which ought to pass after birth from the right ventricle through the lungs to the left ventricle, makes its way directly, *i.e.* by a short cut from ventricle to ventricle through the aperture in the intra-ventricular septum. The blood finds its ways to the lungs either through the ductus arteriosus, or, if as is frequently the case, that vessel is closed, through some branches of the aorta (usually the bronchial, œsophageal, anterior coronary or

¹ *Quain's Anatomy*, ninth edition, vol. ii. p. 875.

pericardial arteries), inosculating with branches of the pulmonary arteries.

When the lesion is produced after the *third* month, the intra-ventricular septum is closed, and compensation is effected by the foramen ovale remaining patent. After birth, the blood, instead of passing from the right ventricle through the lungs to the left heart, passes directly from the right to the left auricle; the lungs are supplied with blood either through the ductus arteriosus remaining patent, or, if that vessel is closed, through the inosculation between branches of the aorta and the pulmonary artery.

In both groups of cases, the compensation is seldom perfect, the superficial veins are usually enlarged, and more or less cyanosis is generally present. The balance of compensation, too, is easily upset, any slight pulmonary affection, for example, causing a great increase in the cyanosis and other symptoms.

The condition of the right ventricle varies in different cases, in some it is markedly hypertrophied; in others, as for instance in those cases in which the orifice is completely occluded, it may be much smaller than normal or quite rudimentary. The lungs are usually found to be anæmic; and in cases which live to the age of puberty, chronic tubercular changes in the lungs are extremely frequent, and are in a large proportion of cases the immediate cause of death.

Clinical History.—It is unnecessary to give a separate description of the acquired and congenital forms of the disease, for the symptoms in acquired cases are identical with the symptoms in some of the congenital cases in which the lesion is not severe, and in which the symptoms do not develop until later life. We may conveniently divide cases of congenital stenosis of the pulmonary artery into three clinical groups or types, viz. :—

First group.—Cases in which the lesion is very severe, and in which the patient dies immediately, or soon after birth.

In the most severe cases of this description, the child dies asphyxiated immediately after birth; in some cases, life is

belonged for a few weeks or months; cyanosis is very prominent, and is very much increased by anything, as for instance coughing or crying, which further embarrasses the lungs or right heart; the temperature is usually subnormal; somnolence is a characteristic symptom, in one case, for instance, which came under my own observation, the child would sleep for eighteen or twenty-four hours at a stretch; dropsy of the feet may develop; shortness of breath and pulmonary complications are often present; convulsions are not uncommon.

Second group.—Cases in which the lesion is less severe, and in which life may be prolonged for several years, but in which there are from the first, symptoms indicative of the cardiac lesion.—In cases of this description there is more or less cyanosis, which may however, only be noticeable on coughing, crying, etc., or when the right heart and pulmonary circulation become embarrassed by any sudden effort, attacks of bronchitis or other lung complications; the blueness is most noticeable in the peripheral parts of the body; the fingers are clubbed; the superficial veins are usually prominent; the child develops slowly, and looks much younger than his years; shortness of breath on exertion, and palpitation are generally prominent symptoms; pulmonary complications, such as attacks of bronchitis and hæmoptysis are frequent; headache, giddiness, or even epileptiform convulsions (the result of deranged cerebral circulation) are sometimes seen. Dropsy is seldom present in cases of this description until the failure of compensation, or unless the venous circulation becomes suddenly much embarrassed by intercurrent complications, such as acute bronchitis, acute endocarditis, etc. Children affected in this manner are very susceptible to cold and all injurious external influences; their mental development may be retarded; and if they survive the trying ordeal of puberty, they are very apt to die in early manhood—between the ages of fifteen and twenty-five—from phthisis; the lung disease, as a rule, runs a protracted course, the left lung is usually the first to be attacked, and repeated hæmorrhage is commonly observed. In exceptional cases

included under this group, life may be prolonged for years. Lebert, for example, mentions that in one case of undoubted congenital stenosis, the patient attained the age of sixty-five years.¹

Third group.—Cases in which the lesion is slight.—In cases of this description, the usual symptoms of congenital heart disease may be slight or entirely absent. Years after birth cyanosis and shortness of breath and the other indications of a right-sided lesion may arise, and are usually due either to the failure of compensation, which has hitherto been perfect, or to the occurrence of acute endocarditis, bronchitis or some other pulmonary complication. When the pulmonary stenosis is acquired the same symptoms may arise.

Physical signs.—In cases of acquired pulmonary stenosis the physical signs are:—(1) A systolic murmur having its point of differential maximum intensity in the second left interspace,

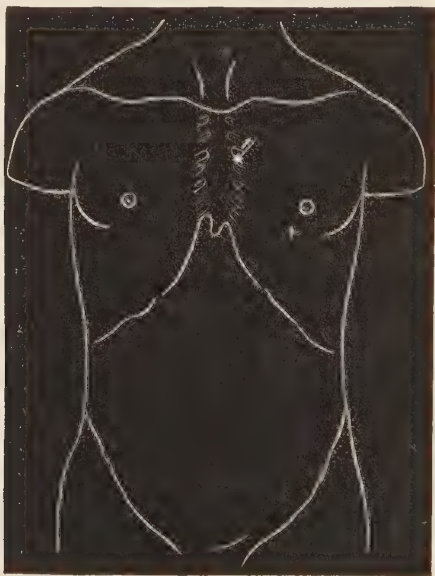


FIG. 230.—Outline figure showing the point of differential maximum intensity of the systolic pulmonary murmur, and the direction in which it is propagated.

¹ Ziemssen's *Cyclopædia*, vol. vi. p. 317.

or at the junction of the third left costal cartilage with the sternum, and its direction of propagation upwards and towards the left (see fig. 230). The murmur, which is in some cases loud and harsh in others soft and faint, is not heard in the carotids; a thrill can in some cases be felt over the position of the murmur. (2) A faint or inaudible pulmonary second sound; in those cases in which the valve is incompetent as well as stenosed, a diastolic, as well as the systolic murmur, is present. (3) Increased dulness over the right heart, and the usual indications of right-sided hypertrophy and dilatation. (4) Fulness of, and in some cases pulsation in, the superficial veins, the external jugulars for example, together with more or less cyanosis. (5) A small and weak, in some cases irregular radial pulse.

In congenital cases, a systolic murmur can generally be heard over the præcordial region; its point of maximum intensity varies in different cases, but is usually situated in the second and third left interspaces; in many cases the murmur is a loud one, and may be heard all over the præcordia. (The exact significance of a systolic murmur in cases of congenital heart disease is not always clear; in some it is probably due to fluid veins formed as the blood passes through the constricted pulmonary orifice; in cases of this description the pulmonary second sound is feeble, altogether absent, or replaced by a diastolic murmur; in others it is probably produced by the passage of the blood through the patent foramen ovale or through an aperture in the intra-ventricular septum; in others, again, it is the result of mitral or tricuspid regurgitation.¹)

A systolic thrill can in some cases be felt over the region of the murmur. When the right ventricle is enlarged, as it is in many cases, increased dulness over the region of the right heart and (in some cases) bulging of the præcordial region are present.²

¹ In this case the murmur would be presystolic rather than systolic.

² The right ventricle is not hypertrophied or dilated in all cases of congenital stenosis of the pulmonary artery. When, for example, the occlusion is complete it may be smaller than normal; when, again, the intra-ventricular septum is imperfect, there may be no alteration in the size of the right heart,

The superficial veins are usually prominent, and in some cases pulsate; the radial pulse is (as a rule) small and weak.

Diagnosis.—When a systolic murmur is heard over the region of the pulmonary artery we have to inquire:—

(1) Is the murmur actually produced in the pulmonary artery?

(2) If the murmur is produced in the pulmonary artery, is it due to functional conditions (such as anæmia) or to organic disease (*i.e.* stenosis of the orifice)?

(3) If organic, is it congenital or acquired?

Further, in all cases of cyanosis and congenital heart disease, whether a systolic pulmonary murmur is present or not, we have to inquire whether the case is one of stenosis or complete occlusion (atresia) of the pulmonary artery.

Step No. 1. Is the murmur produced in the pulmonary artery?—A pulmonary systolic murmur can usually be distinguished from other systolic endocardial murmurs (*viz.*, from systolic aortic, tricuspid, and mitral murmurs), by observing its point of differential maximum intensity, its direction of propagation, the size and shape of the right and left hearts, and the condition of the peripheral venous and arterial circulations. (See Table VIII., p. 527.)¹

Step No. 2. If the murmur is produced in the pulmonary artery, is it due to functional derangement or to organic disease?—Functional (hæmic) murmurs, which are, as we have previously seen, very frequently indeed heard in the pulmonary area, can usually be distinguished from pulmonary murmurs due to organic disease (*i.e.* stenosis of the pulmonary orifice) by attention to the following points:—

1. *The colour of the skin and mucous membranes.*—When the murmur depends on anæmia, the skin and mucous membranes are of course pale, and the other indications of anæmia are present. On the other hand, when the

¹ Naunyn, Balfour, and others, believe that a mitral systolic murmur is sometimes heard in the second left interspace rather than at the apex of the heart. This point is fully considered on p. 190.

murmur is due to organic disease, *i.e.* stenosis of the valvular orifice, the patient is more or less cyanotic.

2. *The nature of the symptoms.*—When the murmur is due to anæmia, lung symptoms and the signs of venous engorgement are not prominent. In organic stenosis, on the contrary, cough, shortness of breath, even when at rest, dropsy, and the other symptoms and signs of venous engorgement, may be very prominent.

3. *The condition of the right heart.*—In anæmia there is usually some enlargement (hypertrophy and dilatation) of the right heart, but it is never extreme; in organic stenosis, on the contrary, the enlargement of the right heart (both ventricle and auricle) may be great.

4. *A venous hum in the neck* is present in anæmia, but (?) not in organic stenosis.

5. *The character of the pulse.*—In the earlier stages of anæmia, the pulse is of good tension, but very irritable and variable in rate; in organic stenosis, the radial pulse is small, weak, and may be intermittent.

6. *The character of the pulmonary second sound.*—Always present and usually accentuated in anæmia; faint, absent or replaced by a diastolic murmur in organic stenosis.

7. *The effects of treatment.*—Anæmia and its attendant pulmonary murmur are, with rare exceptions (some cases of progressive pernicious anæmia) completely and speedily cured by appropriate treatment; organic stenosis is incurable.

This seems an appropriate place to refer to a peculiar loud systolic murmur which is heard over the situation of the pulmonary artery in some cases in which the left lung is retracted. Quincke, who first described this murmur, supposed that it is produced by the pulmonary artery being constricted by the pressure of the heart during the contraction of the ventricles. In the cases in which the murmur is audible, the pulsation of the pulmonary artery can be seen and felt in the second left interspace. A peculiar feature about this murmur is that it disappears during a full inspiration, that is when the pulmonary artery is covered and

separated from the chest wall by the fully expanded lung. Dr G. Balfour, in his Clinical Lectures on the Diseases of the Heart, gives the details of several cases in which a murmur of this description was present, and states that, 'though these cases cannot be held to have proved the correctness of Quincke's theory, they yet lend a very strong support to it.'¹ Two cases, which seemed to be of a similar nature, have come under my own observation, and in them I came to the conclusion that the murmur was exocardial, and produced by the contact of the pulmonary artery, or rather the roughened portion of the pericardium, which covers the root of the pulmonary artery, with the anterior wall of the chest. (In the two cases to which I refer the retraction of the lung was due to pleurisy, and there was every reason to suppose that the outer surface of the pericardium was roughened by a deposit of lymph). 'The very peculiar loudness, roughness, and localised character—not propagated in any direction—of the murmur' (to quote Dr Balfour's description of it) seem to me to lend support to this view; while it is, I think, extremely doubtful whether the heart does or can compress the pulmonary artery in the manner which Quincke has supposed. Recent observations indeed show that the conus arteriosus descends to the plane of the base during the ventricular systole; and that the length of the ventricle does not alter during the contraction of the heart.²

Before leaving the subject of pulmonary murmurs, I must also add, on the authority of Dr Hilton Fagge, and I can from personal observation confirm his statement, 'that in children (even when in good health) a murmur over the pulmonary valves may be generated by the pressure of the stethoscope, as is shown by the fact that it disappears when the instrument is lightly applied. It is said that a similar murmur has sometimes been observed even in adults, when the chest walls are thin and yielding.'³

Step. No. 3.—If the murmur is produced in the pulmonary

¹ *Diseases of the Heart*, p. 225.

² Macalister, *British Medical Journal*, Oct. 28, 1882, p. 822.

³ Russell Reynold's *System of Medicine*, vol. iv. p. 639.

artery, is it congenital or acquired?—It is impossible in some cases to come to a definite conclusion on this point, for, as we have previously seen, it occasionally happens that a congenital lesion of the pulmonary artery remains entirely latent for some years, and is not attended by any symptoms until several, it may be many years, after birth. In cases of this description, it might easily be supposed that the lesion was an acquired one; in fact, in many cases this is actually the fact, as for example in those cases in which the congenitally malformed valve is subsequently attacked by acute or subacute endocarditis. On the other hand, it should be remembered that cases of acquired stenosis of the pulmonary artery are extremely rare. The differential diagnosis of acquired and congenital stenosis of the pulmonary artery is, therefore, to be determined by making a careful inquiry into the history of the case. Cyanosis, shortness of breath, attacks of bronchitis in early life, are very strongly in favour of the congenital variety. The family history should be inquired into, for it is not uncommon to find several members of one family dying from, or affected with congenital heart disease. It is only when the symptoms and signs of cardiac disease have been entirely absent in early life that the diagnosis of acquired pulmonary stenosis can be entertained.

The differential diagnosis of congenital heart disease (e.g. pulmonary stenosis), primary lung disease (e.g. cirrhosis, chronic bronchitis, and emphysema), and mitral stenosis.

All of these conditions may be attended with marked cyanosis, enlargement of the right heart, tricuspid incompetence, and the usual symptoms and signs of systemic venous engorgement; in all of them pulmonary symptoms and signs, and attacks of bronchitis and emphysema may be prominent, and it may require, therefore, great care and discrimination to arrive at a correct conclusion as to the exact nature of the case.

Pulmonary stenosis (*i.e.* congenital heart disease) can usually be differentiated from the other two (*i.e.* primary lung

disease and mitral stenosis) by a careful physical examination and inquiry into the history of the case. In pulmonary stenosis, a pulmonary systolic murmur is usually present, and the second sound is faint, entirely absent, or replaced by a diastolic murmur; whereas in mitral stenosis and primary lung disease, there is no systolic pulmonary murmur, and the second pulmonary sound is loudly accentuated.¹

In the majority of cases of pulmonary stenosis, a history of a shortness of breath and more or less cyanosis from the time of birth can be elicited. (A history of cyanosis and shortness of breath, from early childhood is *not*, however, conclusive evidence of congenital heart disease.) I have known a case of marked cyanosis, with secondary hypertrophy and dilatation of the right heart, and eventually tricuspid regurgitation and dropsy, in which these symptoms dated back from childhood, and in which, therefore, the history of the case was suggestive of congenital heart disease. In cases of this description reliance must chiefly be placed upon the condition of the heart as elicited by physical examination. In cases of congenital heart disease, cyanosis is usually much more prominent than dropsy, whereas in mitral stenosis the reverse is the case. The differential diagnosis of mitral stenosis and of primary lung disease, with secondary changes in the right heart, is in some cases extremely difficult. The manner in which a decision is to be arrived at, has been previously described. (See p. 494.)

Prognosis.—In the great majority of cases of congenital stenosis of the pulmonary artery, the patient dies soon after birth; and even those, who attain the age of puberty, usually succumb during early adult life, tuberculosis of the lungs being a very frequent cause of death. Some few cases live to middle life, and one case has been already referred to, in which the patient reached the advanced age of sixty-five.² The

¹ In complete atresia there may be no pulmonary systolic murmur, but cases of complete occlusion so rarely survive, and even if they do survive, they are attended with such prominent symptoms, continuously from the time of birth, that they could hardly give rise to difficulties in diagnosis.

² Lebert, *Ziemssen's Cyclopædia*, vol. vi. p. 317.

prognosis is, of course, most unfavourable in those cases in which the cyanosis is severe. The acquired form is so extremely rare, that it is hardly possible to lay down any general rule as to the duration and chances of life; each individual case must be judged on its own merits, taking into account the severity of the symptoms, the extent of the secondary derangements in the heart and circulation, and the many other considerations which have been detailed under the head of the various forms of chronic valvular lesions.

Treatment.—Children affected with congenital heart disease must be carefully protected from cold and other injurious external influences; anything which increases the cyanosis (*i.e.* the embarrassment of the right heart and venous circulation, and interferes with the action of the lungs) such as sudden efforts, must be rigidly prohibited; and the therapeutic measures recommended for the treatment of chronic valvular lesions of the left heart, must be carried out in accordance with the requirements of each special case.

PULMONARY INCOMPETENCE.

Ætiology.—Pulmonary regurgitation is extremely rare: it is occasionally met with as a congenital condition, and is then generally combined with pulmonary stenosis. In extremely rare cases, pulmonary incompetence is produced after birth in the course of ulcerative endocarditis. It hardly ever results from the simple form of endocarditis or atheromatous changes. Theoretically we might suppose that ‘relative’ incompetence of the pulmonary valves might be caused by over-distention and dilatation of the pulmonary artery, but, as a matter of fact, the dilatation of the vessel is seldom, if ever, sufficiently great to interfere with the perfect closure of the valve flaps.

Pathological physiology.—The effect of pulmonary regurgitation would, of course, be to allow a certain quantity of blood, which ought to be retained in the pulmonary artery and lungs, to pass backwards into the cavity of the right

ventricle during the ventricular diastole. As in aortic incompetence, hypertrophy and more especially dilatation of the right ventricle, would result; this would ultimately be followed by relative incompetence of the tricuspid, and the usual symptoms and signs of systemic venous engorgement. In congenital cases, the incompetence is usually, as I have already mentioned, combined with stenosis; and in cases of this description, the foramen ovale remains open, and the extreme engorgement of the systemic venous circulation is thereby relieved.

Clinical History.—In congenital cases, the symptoms are identical with those of pulmonary stenosis. In those cases in which the lesion is due to ulcerative endocarditis, symptoms and signs of rapid embarrassment of the right heart, of embolic infarctions in the lungs and other pulmonary lesions, and of engorgement of the systemic venous circulation, together with the grave constitutional symptoms which characterise ulcerative endocarditis (see page 406) would be present. In those infinitely rare cases, in which pulmonary regurgitation is slowly and gradually established after birth, secondary changes, more especially hypertrophy of the right ventricle, may for a time compensate the lesion. Shortness of breath, cough, and other indications of pulmonary derangement would probably be present through the stage of compensation. Sooner or later the tricuspid would give way, and the symptoms due to engorgement of the venous systemic circulation would develop.

Physical signs.—Pulmonary incompetence is attended with a diastolic murmur, which has its point of differential maximum intensity in the pulmonary area, and its direction of propagation downwards and towards the right. The murmur, like the diastolic murmur of aortic regurgitation, would probably in many cases be best heard at the lower end of the sternum. The usual physical signs indicative of hypertrophy and dilatation of the right heart are also present. The venous system is engorged, the arterial system empty, the pulse being small, weak, and probably irregular.

Diagnosis.—The diastolic murmur indicative of pulmonary incompetence might be readily mistaken for the diastolic murmur of aortic regurgitation, since the latter is so common, the former so rare. The points of distinction between the two conditions are given in the following table:—

TABLE IX. — *The Differential Diagnosis of the Diastolic of Aortic Murmurs and Pulmonary Incompetence.*

	Aortic Incompetence.	Pulmonary Incompetence.
<i>Point of differential maximum intensity of the murmur.</i>	Second right costal cartilage.	Second left interspace, or third left cartilage.
<i>Direction of propagation of murmur.</i>	Downwards and to the left.	Downwards and to the right.
<i>Condition of left ventricle.</i>	Dilated and hypertrophied.	Normal (unless some complication).
<i>Condition of right ventricle.</i>	Normal (unless some complication, such as secondary mitral incompetence.)	Hypertrophied and dilated.
<i>Condition of radial and other superficial pulses.</i>	Jerking, visible, collapsing, tortuous.	Small, weak, and often irregular.
<i>Condition of venous system.</i>	Normal (unless some complication such as secondary mitral incompetence).	Engorged, often extremely so, and all the usual results of venous obstruction, such as dropsy, present.
<i>Condition of pulmonary circulation.</i>	Normal (unless some complication, such as secondary mitral incompetence).	Distended during the ventricular systole, empty during diastole; pulmonary symptoms usually prominent.

Prognosis.—Unfavourable as regards the ultimate result, though Rosenstein states that ‘if we may draw conclusions from the exceedingly limited number of cases on record, the length of the patient’s life . . . may be comparatively long.’¹

Treatment.—In the earlier stages of the case, the same treatment which has been recommended for aortic regurgitation (see 519) should be adopted; after venous engorgement becomes prominent the treatment suitable for mitral lesions is to be employed.

¹ *Ziemssen’s Cyclopaedia*, vol. vi. p. 155.

CHAPTER VI.

DISEASES OF THE MYOCARDIUM. ACUTE MYOCARDITIS. CHRONIC MYOCARDITIS OR FIBROID DEGENERATION. PARTIAL ANEURISMS OF THE HEART. HYPERTROPHY AND DILATATION. HYPERTROPHY OF THE LEFT VENTRICLE. HYPERTROPHY OF THE RIGHT VENTRICLE. HYPERTROPHY OF THE AURICLES. ATROPHY OF THE HEART. FATTY INFILTRATION. FATTY DEGENERATION. SPONTANEOUS RUPTURE. TUMOURS OF THE HEART.

THE diseases of the myocardium or muscular wall of the heart, as distinct from the pericardium and endocardium, include a variety of different conditions. Those which are of practical and clinical importance, and which we shall therefore, consider in detail, are :—

1. The inflammatory affections and their results (acute and chronic myocarditis, fibroid degeneration, chronic aneurismal dilatation of the cardiac cavities).
2. Hypertrophy, dilatation, and atrophy of the heart.
3. The various forms of muscular degeneration, amongst which the fatty form is of the most importance from a practical and clinical point of view.

The new growths of the heart, which form a fourth group, are extremely rare, and are of more interest to the pathologist than the practical physician.

ACUTE MYOCARDITIS.

Definition.—Acute inflammation of the muscular wall of the heart.

There is considerable difference of opinion as to what should and what should not be included under the head of acute myocarditis. Some authorities consider that the changes which are met with in the muscular fibres of the heart in certain febrile affections, notably in typhus and typhoid, are inflammatory; and Virchow indeed divides cases of myocarditis into two groups, viz., the *parenchymatous* and the

interstitial; other observers doubt the occurrence of a purely parenchymatous inflammation of the cardiac wall, and think, that in all cases of myocarditis, alterations are to be detected in the fibrous septa and blood vessels, which lie between the muscular fibres, as well as in the muscular tissue itself. Personally I agree with the latter view, and I am, with Gowers,¹ in the habit of including cases of so called parenchymatous myocarditis under the head of degenerative rather than inflammatory changes. I do not, of course, mean to imply that myocarditis is never observed in typhoid and typhus, but only that the ordinary changes (cloudy swelling and acute fatty changes) which occur in these affections, and indeed in all cases of continued pyrexia, are not in my opinion of an inflammatory nature.

Ætiology.—Acute myocarditis is very rarely primary, but usually arises in the course of some general affection. Acute articular rheumatism is the disease *par excellence* with which it is most frequently associated; in the majority of these cases (*i.e.* of rheumatic myocarditis) the inflammatory process seems to arise in the pericardium or in the endocardium, and to extend subsequently to the muscular substance of the heart. In some rheumatic cases the inflammation is probably limited to the myocardium. Inflammation of the myocardium is occasionally met with in pyæmia and puerperal fever, and in cases of this description secondary abscesses may form in the wall of the heart. New growths, which are of rare occurrence in the heart, may produce inflammation of the muscular tissue which surrounds them. Traumatic injuries seem occasionally to be the cause of acute myocarditis. Embolic plugging of branches of the coronary artery also seems in some cases to be followed by changes of an inflammatory character in the cardiac wall.

Pathology.—In some cases, the inflammation is general or involves extensive portions of the wall of the heart; in others, it is local or limited in distribution. The ventricles are more

¹ Russell Reynolds' *System of Medicine*, vol. iv. p. 529.

frequently affected than the auricles. After birth the left ventricle is much more liable to be affected than the right; during intra-uterine life the contrary holds good. The apex of the left ventricle seems to be the part which is especially liable to be attacked, then the septum ventriculorum, then the posterior wall of the organ.

On naked eye examination, the affected portion of the heart is, in the earlier stages, of a dark purple or bright red colour, the result of the increased quantity of blood which it contains; in the later stages it may be paler than in health. The consistency of the muscular tissue, more especially in the later stages of the affection, is softer than normal. According to some authorities (Stein quoted by Schroetter¹) the myocardium is sometimes studded throughout with spots of albuminous exudation, which have a glistening appearance, and resemble little deposits of fat scattered through the muscular tissue; personally I have not, so far as I can remember, observed this change.

On microscopical examination, the vessels of the myocardium are seen to be dilated and engorged; the fibrous septa between the muscular fibres and around the blood vessels are more or less infiltrated with cellular elements (leucocytes, red blood corpuscles, and proliferating nuclei of the cellular tissue, see figs. 233 and 234²), and in some rare cases (purulent myocarditis) well marked collections of pus—small abscesses—may be detected under the pericardium or in the substance of the wall of the heart. The muscular elements may be little altered, but in typical examples of acute myocarditis they are swollen, and their normal striation is indistinct or completely absent in consequence of cloudy swelling, glassy

¹ *Ziemssen's Cyclopadia*, vol. vi. p. 228.

² In the preparation from which figures 233 and 234 was drawn, the fibrous septa were infiltrated with exudation products and large collections of leucocytes were present here and there in the deeper layer of the pericardium. The muscular fibres were for the most part quite healthy. The section was made from an advanced case of valvular disease. In typical cases of acute myocarditis, the same changes which are present in the perimysium in this case can also be observed, but in addition the muscular fibres themselves are (?) always more or less affected in the manner described in the text.

FIG. 231.

FIG. 231'.

FIG. 232.

FIG. 233.

FIG. 231.—Muscular fibres in sub-acute myocarditis, showing great enlargement of their nuclei ($\times 300$).

FIG. 231'.—Muscular fibres in chronic myocarditis ($\times 250$).

a, a, muscular fibres transversely divided, showing vacuoles; *b, b*, the same in longitudinal section; *c, c*, nuclei within the vacuolated fibres.

FIG. 232.—Muscular fibres in chronic myocarditis ($\times 250$).

The fibres are swollen; their transverse striæ have almost completely disappeared; the fibres are granular, but not fatty.

FIG. 233.—Section through the wall of the left ventricle in a case of chronic mitral disease (\times about 200).

The fibrous tissue between the muscular fibres is increased, and infiltrated with leucocytes and red blood corpuscles; the muscular fibres are healthy. *m, m*, muscular fibres; *f, f*, fibrous tissue; *c, c*, connective tissue corpuscles; *l, l*, leucocytes; *r, r*, red blood-corpuscles; *v*, blood-vessel.

degeneration, or fatty changes; the affected fibres are more brittle than usual, and tend to split up transversely into short fragments. In some cases, more especially in purulent myocarditis, the muscular fibres at the seat of the inflammation may be entirely destroyed, and a mass of broken down tissue, consisting of granular debris, fatty particles, leucocytes, and a few red blood corpuscles, may be all that remains. In sub-acute myocarditis, the nuclei of the muscular fibres are often enormously enlarged. (See figs. 231 and 237.)

Pathological physiology.—The effects produced by myocarditis vary very considerably in different cases, and depend upon the severity, extent, and character of the inflammation. When extensive portions of the muscular wall of the heart are implicated, the force of the cardiac pump is seriously impaired, and acute dilatation of the affected cavities may result; regurgitation due to ‘muscular’ and ‘relative’ incompetence of the mitral or tricuspid orifices also occurs in cases of this description. Auriculo-ventricular regurgitation may also result from a local inflammation of the muscular fibres surrounding the mitral or tricuspid orifices, the other parts of the cardiac wall being healthy. Acute cardiac aneurisms, *i.e.* acute local bulgings of some part of the cardiac wall, and rupture of the heart are occasionally met with as the result of inflammation and softening, limited to some particular part of the wall of the organ. When an abscess forms it may make its way either externally into the pericardium, producing acute purulent pericarditis, or internally through the endocardium; in the latter case, which is probably of very rare occurrence, the contents of the abscess escape into the circulation, and may add to the septic (pyæmic) symptoms, which are usually present in cases of this description. (Purulent myocarditis is rarely met with except in cases of pyæmia.) When the abscess occupies the septum ventriculorum, perforation of the septum may be produced, and a communication established between the two ventricular cavities. Communication between the two auricles might be produced in a similar manner, *i.e.* by the bursting of an abscess, the result of ulcerative endocarditis in the auricular septum.

Symptoms and physical signs.—The symptoms and physical signs of acute myocarditis are for the most part very indefinite. In the slighter forms of the disease, such as are, I believe, of much more frequent occurrence in acute rheumatism than is generally supposed, symptoms and signs indicative of myocarditis may be entirely absent or extremely slight. In more severe cases, the patient usually complains of some uneasiness, and occasionally of actual pain in the region of the heart; a feeling of tightness, shortness of breath, and palpitation are frequently experienced. There is, too, more or less (often extreme) exhaustion, languor, and debility, and frequently a short dry cough. In severe cases, the signs of venous engorgement and stasis, such as have been described under the head of acute endocarditis and chronic valvular lesions, are present. The pulse is considerably quicker than normal; in the earlier stages of the disease it may be unusually irritable; in the latter stages it is small, and often very irregular. There is generally some pyrexia; the increased temperature partly depending upon the myocarditis, but being for the most part due to the primary affection with which the myocarditis is associated,—acute rheumatism for example—or to associated pericarditis or endocarditis. In cases of purulent myocarditis, a suppurative temperature, characterised by frequent ups and downs, and associated with rigors and sweatings, is usually observed.

On examining the heart, the physical signs of pericarditis, or of endocarditis, which are so generally associated with acute myocarditis can usually be detected. In uncomplicated cases, *i.e.* where there is no associated pericarditis or endocarditis, the action of the heart may, in the earlier stages, be unusually irritable. Soon, however, the signs of cardiac failure, dilatation and auriculo-ventricular regurgitation become apparent. The impulse becomes weak or altogether imperceptible; the area of dulness more or less increased; the first sound short and faint, or replaced in the mitral and tricuspid areas, by a soft blowing murmur.

Diagnosis.—The diagnosis is in many cases difficult or

impossible. Acute myocarditis may be suspected, when in addition to more or less pyrexia, the symptoms and signs of cardiac failure (perhaps preceded by indications of cardiac irritability) are acutely developed, in the course of a disease, such as acute rheumatism, in which inflammation of the myocardium is apt to occur. A positive diagnosis of acute myocarditis can only be given, when, in addition, the observer feels satisfied that the symptoms and signs do not depend upon pericarditis and endocarditis. (Pericarditis and endocarditis are frequently complicated, as we have already seen, with inflammation of the muscular layer of the heart; and in cases of acute pericarditis and acute endocarditis it is often extremely difficult to determine what proportion of the symptoms, so to speak, depends upon the inflammation of the pericardium, of the endocardium, and of the myocardium respectively.)

The differential diagnosis of acute endocarditis and acute myocarditis is especially difficult, and in many cases impossible. It may be impossible, for example, in a case of rheumatic fever, in which mitral regurgitation is developed at an early stage of the case, to say whether the incompetence is 'muscular' and due to myocarditis, or whether it depends upon endocarditis. But into the details of this question, which have been discussed in treating of acute endocarditis (see p. 373) I need not again enter.

The presence of purulent myocarditis may be suspected, when the symptoms and signs of cardiac weakness and irritability are acutely developed in the course of pyæmia or other conditions, in which purulent myocarditis is apt to arise. If in addition acute pericarditis is very quickly developed, or, if symptoms of arterial pyæmia rapidly arise, the observer may suspect that an abscess in the myocardium has ruptured into the pericardium or into the interior of the heart. It is seldom possible to do more in the way of diagnosis than suspect these conditions; there are many fallacies which deter a careful and thoughtful investigator from committing himself to a positive opinion in cases of this description, though the imperfectly informed, and often plausible physician who has no difficulty in giving a sharply defined diagnosis in any and

every case (and who, it may be observed is, often unable to give the grounds for his opinion, or to appreciate correctly the arguments which may be brought against it) may occasionally make a happy hit.

Prognosis.—Acute myocarditis, in its more severe and pronounced forms, is a very serious affection, and adds, as we have previously seen, very materially to the danger of any other cardiac affection. Slight degrees are, I think, of much more frequent occurrence in acute rheumatism than is generally supposed, and are, I believe, frequently recovered from. In attempting to gauge the gravity of each individual case, the amount of cardiac weakness (which is determined by observing the force of the cardiac impulse; the extent of the cardiac dilatation; the character of the first sound; the severity of the symptoms indicative of mechanical derangement of the venous circulation; and especially the condition of the radial pulse), is the point which must be chiefly relied upon. It must, however, be remembered in considering the prognosis, that a localised inflammation of the cardiac muscle, insufficient to produce any very distinct or marked symptoms and signs, may produce such an amount of softening of the cardiac wall as may result in the formation of an acute aneurism or may cause rupture of the organ. Localised myocarditis leading to such complete softening as is likely to produce acute aneurism or rupture of the heart, is seldom seen in rheumatic cases. It is more likely to be produced in the purulent myocarditis, or as the result of the embolic plugging of the coronary artery or its branches. Purulent myocarditis is almost certainly fatal.

Treatment.—The same general measures which have been recommended in the treatment of acute pericarditis and acute endocarditis,¹ must be adopted, the greatest care being taken to avoid everything which is likely either to excite or depress the action of the heart. The main object of treatment (for

¹ The reader is recommended to refer to the treatment of acute pericarditis and acute endocarditis (see pp. 336 and 337).

we know of no means of cutting short and arresting the cardiac inflammation) is to sustain the strength of the heart, and at the same time to relieve the organ from all strain, and to keep it as tranquil as possible.

In rheumatic cases, salicylate of soda, which exerts a decidedly depressing effect upon the organ, must, as soon as there is reason to suspect the onset of acute myocarditis, be at once discontinued, though, according to Dr Maclagan, salicin itself may still be given. Although I am quite satisfied that salicylate of soda is more depressing than salicin, I prefer to err on the side of caution. In cases, then, of acute rheumatism, in which there is distinct evidence of myocarditis, I would recommend that not only salicylate of soda, but also that salicin be discontinued, and that quinine, or quinine and bicarbonate of potash, be substituted for these drugs. The patient must be kept at perfect rest in bed, and all sources of agitation or excitement rigidly avoided. During the stage of cardiac excitement and irritability, which is often observed at the commencement of the attack, belladonna and digitalis are the most useful drugs. During the subsequent stages of the disease, small doses of digitalis and quinine may be given. In the treatment of acute myocarditis, it is very important to look out for symptoms and signs of cardiac failure, and when necessary to strengthen and stimulate the action of the heart. One of the greatest dangers is failure of the heart's action, and it is absolutely necessary in all cases in which indications of cardiac failure arise (notwithstanding the risk of producing rupture in those cases in which advanced localised softenings are present) to administer freely, alcoholic and ammoniacal stimulants, ether, and digitalis. Other symptoms and complications, such as pain over the præcordia, shortness of breath, cough, dropsy, embolic infarctions, etc., must be treated by appropriate remedies. In septic cases, and in cases of suspected purulent myocarditis, the measures which have been recommended for the treatment of acute ulcerative endocarditis may be employed.

CHRONIC MYOCARDITIS.

Synonym.—Fibroid degeneration of the heart.

Definition.—Chronic inflammation of the muscular wall of the heart. In this condition, which for practical purposes may be considered synonymous with fibroid degeneration of the heart, bands of fibrous tissue are developed in the wall of the heart and around the muscular fibres, which become atrophied, as will be presently described.

Ætiology and Pathology.—Chronic myocarditis or fibroid degeneration of the heart, is, in many cases, the direct result of a previous attack of acute rheumatic myocarditis. In cases of this description, the pericardium and endocardium are usually at the same time implicated, and chronic thickening of one or other, or both, of these structures is found after death. It is generally supposed that the myocarditis, which is the starting point of the fibroid change, is secondary to an inflammation of the pericardium or endocardium. Possibly in some cases, as Dr Hilton Fagge supposed, the order of events is reversed, and the thickening of the pericardium and endocardium is the result of a fibroid change commencing in the myocardium. In other cases of rheumatic origin (but these are much more rare), the myocardium is alone affected, the pericardium and endocardium being healthy. It is interesting to observe that in most cases of fibroid degeneration, even where the whole thickness of the heart wall is involved, a narrow band of muscular fibres usually remains just internal to the endocardium. This appearance is well seen in figs. 176, 336, and 245. In the preparation, represented in fig. 176, the endocardium and pericardium are enormously thickened; in that case, in which endocarditis, pericarditis, and myocarditis were present, the condition was of rheumatic origin. In the preparation shown in fig. 245, the pericardium and endocardium are healthy; in the latter case the condition was probably of syphilitic origin.

In some cases, the fibroid change is slowly and gradually developed, and is chronic from the first. Some of these cases

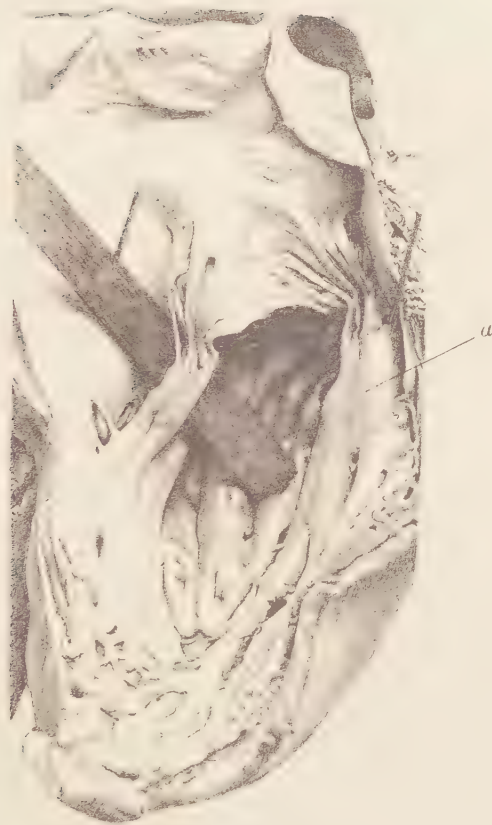


FIG. 235. — *Fibroid degeneration of the heart, showing atrophy of one of the papillary muscles.*
(Natural size.)

A piece of whalebone has been placed beneath the atrophied papillary muscle; its chordæ tendinæ are somewhat thickened and retracted. The letter *a*, points to the opposite papillary which is healthy.

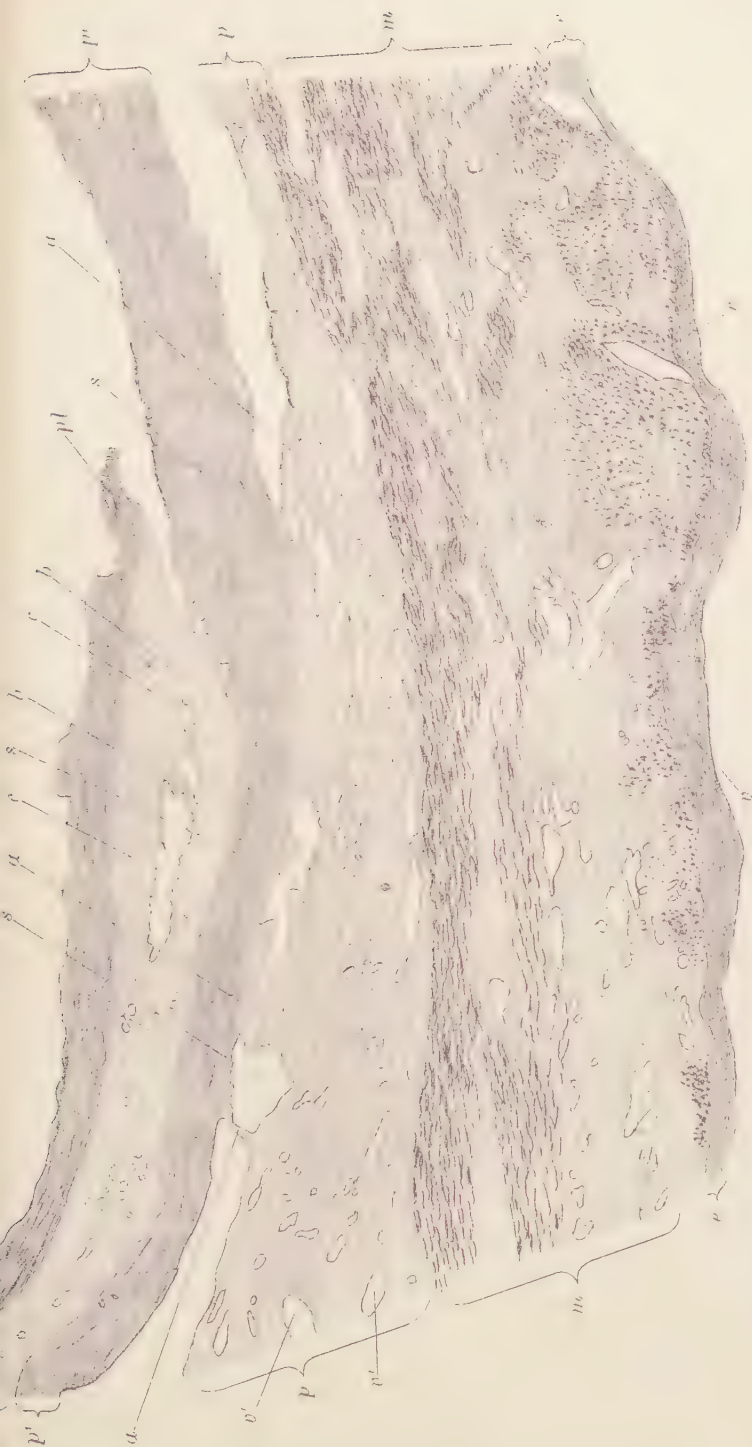


FIG. 236.--Section through the wall of the right auricle, pericardium and pleura, in a case of *endo-peri-myocarditis*. (Magnified about 10 diameters.)

e, e, thickened endocardium; *m, m*, myocardium which is extensively affected with fibroid degeneration; *p, p*, visceral layer of the pericardium; *p', p'*, parietal layer of the pericardium; *pl, pl*, pleura; *a, a, a*, adhesions passing between the two layers of the pericardium (*p* and *p'*); *b, b*, adhesions passing between the outer surface of the pericardium and the pleura (*p'* and *pl*); *s, s*, spaces between the pericardial adhesions; *c, c*, spaces between the pericardium and pleural adhesions; *v, v*, large blood-vessels in the wall of the heart; *v', v'*, blood-vessels in the thickened pericardium.

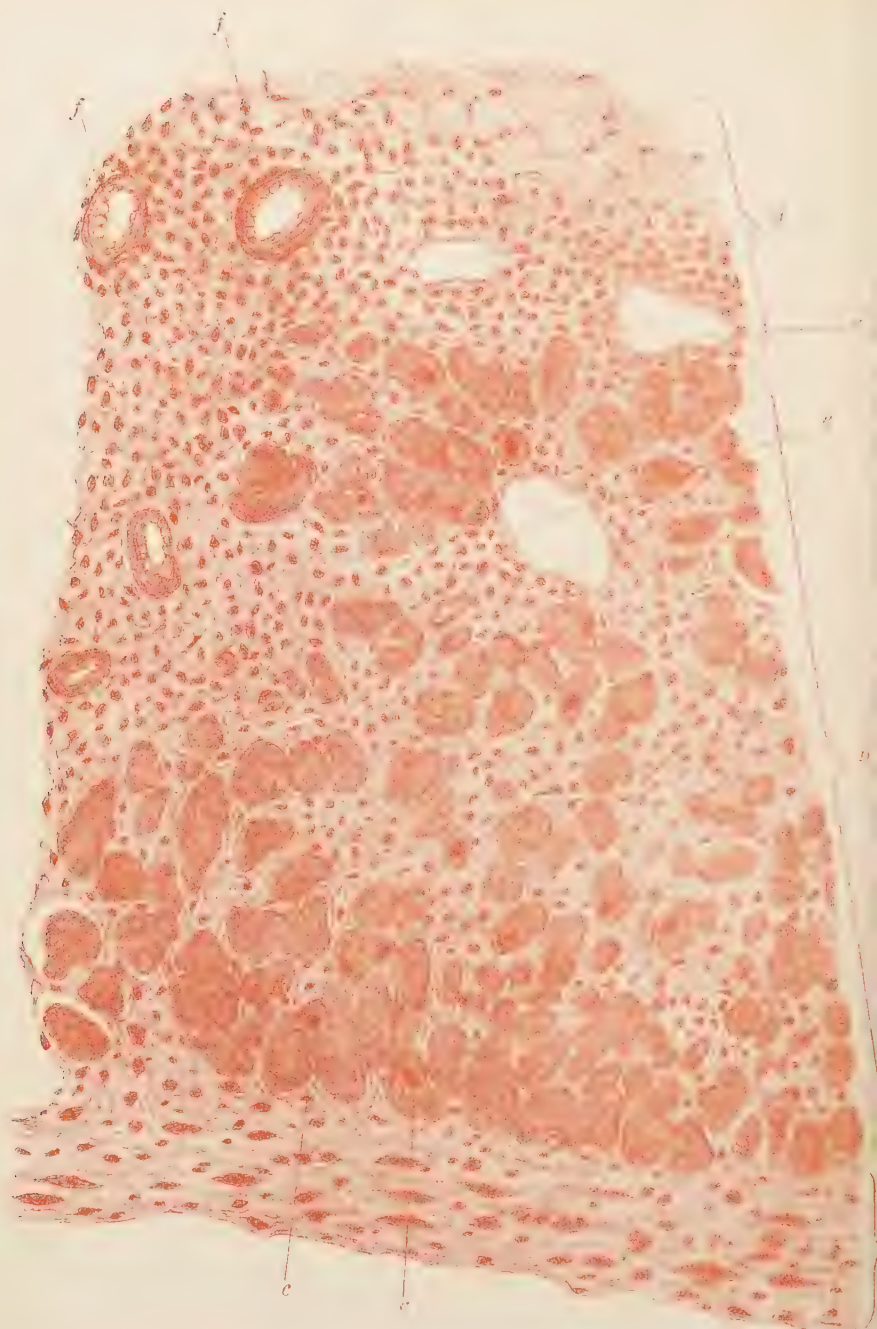


FIG. 237. - Section through the endocardium and adjacent part of the wall of the left ventricle in endomyocarditis. (Magnified about 300 diameters.)

a, thickened endocardium containing numerous corpuscles and spindle cells. *b*, a band of muscular fibres which still remains adjacent to the endocardium; some of the muscular fibres contain large nuclei (*c, c*). An extensive tract of fibroid tissue (the endocardial end, *d*, can only be seen in this figure), is situated in the wall of the left ventricle; it contains numerous corpuscles and nuclei *e, e*, veins surrounded by fibroid tissue. *f, f*, arteries, the walls of which are very thick. (The coronary arteries were very atrophied; in one part of the section, from which this drawing was made, a hemorrhage, probably due to rupture of a branch of the coronary artery, was seen.)



FIG. 238.—Section through the wall of the left ventricle in chronic myocarditis, showing numerous blood-vessels and the remains of muscular fibres in the midst of the fibrous tissue. (Magnified about 250 diameters.)

a, fibrous tissue; *b*, blood-vessel; *c*, *c*, atrophied muscular fibres; *d*, leucocyte, and *e*, nucleus of fibrous tissue.

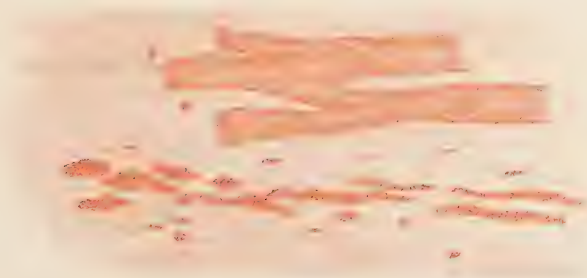


FIG. 239.—Another portion of the same preparation showing healthy and atrophied muscular fibres in the midst of fibrous tissue. (Magnified about 300 diameters.)

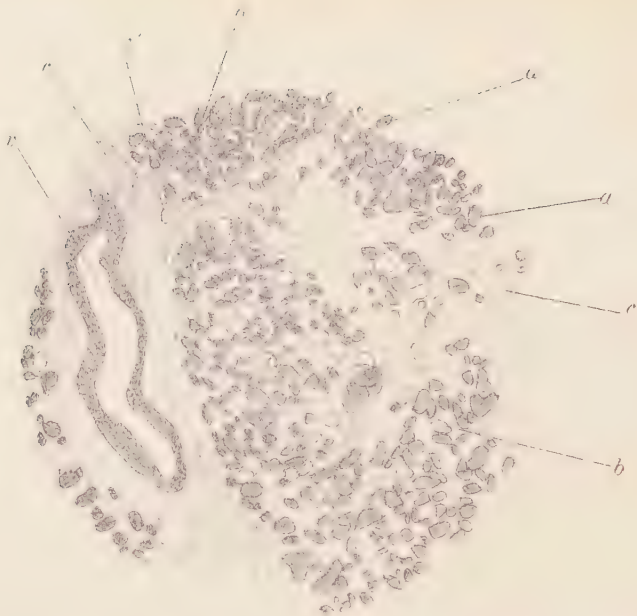


FIG. 240.—Section through the wall of the left ventricle in chronic myocarditis. (Magnified about 50 diameters.)

a, a, muscular fibres transversely divided; *b, b*, empty spaces from which the muscular fibres have fallen out; *c, c*, fibrous tissue between the muscular fibres; *v*, large, and *v'*, small blood-vessels transversely divided.

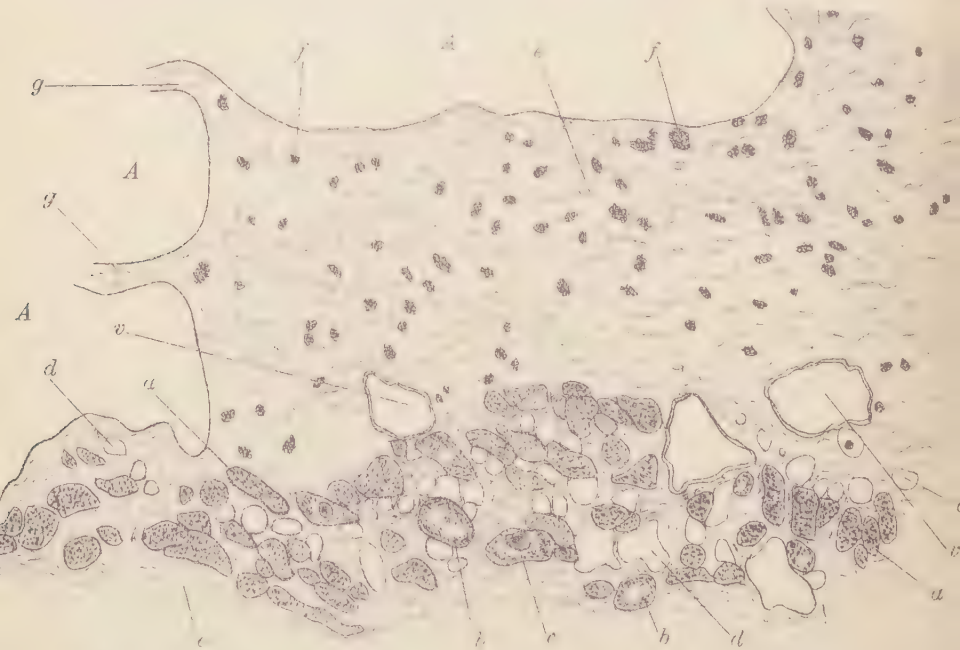


FIG. 241.—Section through the wall of the left ventricle in chronic myocarditis. (Magnified about 250 diameters.)

a, a, healthy muscular fibres transversely divided; *d, d*, vacuolated muscular fibres, some of them containing nuclei; *e, e, d, d*, empty spaces from which the muscular fibres have fallen out; *e, e*, fibrous tissue; *f, f*, the remnants of pigment in muscular fibres (some of which are mere collections of pigment); *v, v*, blood-vessels; *g, g*, the walls of large spaces (containing blood) situated in the midst of the fibrous tissue. The letters *A, A, A*, are placed in these spaces.

seem also to be of rheumatic origin ; others are undoubtedly due to syphilis. Long continued alcoholic excess is probably another cause of the condition. In a considerable proportion of cases of granular kidney, the hypertrophied heart (which is developed in that condition unless the organ is so damaged by previous disease as to be incapable of becoming hypertrophied) presents, on microscopical examination, well-marked fibroid changes. Probably in some cases of this description, the cirrhosis of the kidney and the cirrhosis or fibroid degeneration of the heart are both due to chronic alcoholism. The heart, represented in fig. 235, from which the section shown in fig. 240 was made, was a typical example of fibroid degeneration, the papillary muscles in particular being extremely atrophied. The patient was an old woman who for years had indulged freely in whisky, and who had never, so far as I could ascertain, suffered from rheumatism. The kidneys were typically cirrhotic, and yet the heart was not hypertrophied. Possibly in this case the fibroid degeneration of the heart was the first event, the kidney lesion followed, and owing to the damaged condition of the cardiac muscle, hypertrophy of the left ventricle could not occur. The fibroid lesion of the cardiac wall was very probably in this case (in some measure at all events) the result of alcohol.

Long continued venous engorgement of the wall of the heart, such as results from mitral disease, may also lead to the production of fibroid changes between the muscular fibres, just as it may lead to a form of cirrhosis of the liver or of the kidney ; indeed, as has been pointed out by Dr Charlewood Turner, the perimysium, or fibroid tissue around the muscular fibres of the heart, is increased in many cases of hypertrophy resulting from valvular disease. Dr Turner thinks that the perimysium is thickened in the great majority of cases of hypertrophy of the heart whether the hypertrophy result from chronic Bright's disease or from valvular lesions. I, too, have found changes similar to those which he has described in a considerable number of cases, but my observations do not as yet warrant me in believing that this fibroid change in the myocardium is so frequently present as Dr Turner seems to suppose.

Traumatic injuries in some cases seem to have been the exciting cause of the condition. In pseudo-hypertrophic paralysis, fibroid patches are sometimes seen in the wall of the heart, and undoubtedly owe their origin to the causes which produced the fibroid and atrophic changes in the voluntary muscle.

Males are probably more frequently affected with fibroid degeneration of the heart than females. The condition is seldom met with in young subjects, except as the result of rheumatic causes or pseudo-hypertrophic paralysis; it is much more common after than before thirty years of age.

In some cases the fibroid change is uniformly distributed through large portions, or through the whole, of the organ; and in cases of this description, it probably owes its origin to long continued venous engorgement or chronic alcoholism. In others, the fibroid change is much more marked in, or is confined to, limited portions of the wall of the heart. In intra-uterine life the right ventricle is the part which is said to be chiefly affected. After birth the left ventricle and the septum ventriculorum are the parts which are chiefly attacked, but my experience does not altogether agree with those who believe that the fibroid change is usually confined to the cavity of the left ventricle. I admit, however, that the lesion is, as a rule, more extensive and more advanced in that cavity than in the other parts of the organ.

The naked eye appearances differ in accordance with the stage of the lesion and the distribution of the fibroid tissue. When the whole thickness of the heart is involved, and the fibroid change consists in a thickening of the perimysium around individual fibres (such as is shown in fig. 240) rather than the production of large masses of fibroid tissue, there is little or no alteration visible to the naked eye; the heart perhaps looks hypertrophied; its colour is perhaps paler than normal, and its consistency somewhat firmer than natural. When, on the contrary, the fibroid change is more localised and large masses of new tissue are formed between and around the muscular fibres (see figs. 176 and 236), the naked eye changes may be very visible; the masses of fibroid tissue being of a yellow or

yellowish white colour stand out prominently, usually in the form of streaks or bands between the red or reddish-brown masses of muscle. The wall of the heart at the affected part is tougher than normal, in some cases it is almost cartilaginous in hardness, in others it cuts 'gritty,' owing to the deposit of calcareous particles in the midst of the fibroid tissue.

It not unfrequently happens that the adjacent parts of the pericardium or endocardium are considerably thickened, and the thickness of the cardiac wall, taken as a whole, is increased. (See fig. 236.) In other cases, the wall of the heart at the seat of the lesion is thinner than usual; and should it be, as it often is, at the same time dilated, a local bulging or aneurismal dilatation may be formed at the affected spot. (See fig. 245.) The papillary muscles are the parts which are most frequently affected by fibroid degeneration; they look shrunken, flattened, and atrophied; they lose their normal reddish colour, and become pale and white; the chordæ tendineæ may be thickened and retracted.

On microscopical examination, masses of fibroid tissue which stain of a bright pink colour with picro-carmin, are seen between and around the muscular fibres. In those cases in which the fibroid change is diffused throughout the heart, the individual muscular fibre may be surrounded with a band of fibrous tissue (see fig. 240), which in recent cases (in sub-acute myocarditis) is rich in nuclei and spindle-celled elements. (See fig. 237.) In other cases, and more particularly in those cases in which the fibroid change is localised in one particular part of the heart, large masses of fibroid tissue are placed between masses of muscular tissue. (See figs. 176 and 236.) In cases of this description, the fibroid patches consist, for the most part, of wavy bundles of white fibrous tissue which stain pink with picro-carmin. In other and rarer cases,—and these, so far as my experience goes, seem to be chiefly syphilitic,—the new growth contains large quantities of extremely fine elastic fibres which stain yellow with picro-carmin.

The masses of fibroid tissue are richly supplied with blood; indeed, in some cases, enormous vessels are found in the midst of these fibroid growths. The muscular fibres, surrounded by

and adjacent to the fibroid growth, undergo a gradual process of atrophy, and may entirely disappear, their former position being perhaps indicated by small masses of granular yellow pigment. In this process of atrophy, as Dr Charlewood Turner has so well described, and as many of my preparations beautifully demonstrate, the fibrillæ disappear from the central parts of the fibres, leaving well defined spaces, in which large nuclei are sometimes seen. (See *c.* fig. 231'.)

In some cases, and, so far as my experience teaches, in syphilitic cases, the arteries in the neighbourhood, or in the midst of the fibroid patches, may be narrowed by endarteritis obliterans. (See figs. 245 and 273.)

In pseudo-hypertrophic paralysis, judging from one case which I have been able to examine carefully, the fibroid tissue seems, in part at least, to be derived from the muscular fibres themselves. In the acute and subacute forms of myocarditis, some of the muscular fibres are usually found in a condition of fatty degeneration. In the more chronic forms, such as are commonly classed under the head of fibroid degeneration, fatty degeneration of the muscular elements is not generally seen, but the muscular fibres seem to disappear by a process of simple atrophy; ultimately they may completely disappear, their place being represented by a few pigment granules. (See figs. 238, 239, and 240.)

Pathological Physiology.—The effect of chronic myocarditis, or fibroid degeneration of the heart, as we may term it, is to produce atrophy of the proper muscular elements, and therefore to impair the force of the cardiac pump, and the resistance which the cardiac walls naturally oppose to the blood pressure. The effects, of course, vary with the extent of the lesion, and the cavity or cavities which happen to be affected.

When the fibroid change is pretty generally diffused—through the wall of the left ventricle, for example—there is a tendency to general dilatation of that cavity; the driving power of the left ventricle is decreased, less blood is consequently expelled into the arterial system than under normal circumstances, and blood tends to stagnate in the parts of the circulation behind the affected cavity, *i.e.* behind the

cavity of the left ventricle; defective muscular closure, *i.e.* 'muscular' incompetence, at the mitral orifice, may then be established.

When, on the contrary, the fibroid change is limited to one particular part of the heart, say to the wall of the left ventricle in the neighbourhood of the apex, as in fact it often is, the driving power of the left ventricle is little if at all impaired, there is little or no engorgement of the circulation behind, no 'muscular' incompetence at the mitral orifice; the resisting power of the ventricular wall at the seat of the lesion is, however, weakened, and a local bulging or aneurismal dilatation, which in some cases ruptures and causes sudden or instantaneous death, may be produced.

When again the papillary muscles are affected and atrophied, the perfect working of the mitral valve is apt to be interfered with, and mitral incompetence produced.

In some cases, the fibroid mass instead of dilating under the pressure of the blood gradually contracts, just as an external cicatrix (a cicatrix in the skin, for example) does. Stenosis of the conus arteriosus (true stenosis of the heart), a condition to which I have alluded in speaking of stenosis of the pulmonary artery, seems to be due to the contraction of a circular band of fibrous tissue—the result of foetal endocarditis—developed all round the wall of the conus.

Symptoms and physical signs.—The symptoms and physical signs of chronic myocarditis are even more obscure and indefinite than those of the acute form.

The diffuse variety is characterised by the usual symptoms and indications of failure of the cardiac pump, *viz.*, shortness of breath, in some cases only felt on exertion, in others (*i.e.* in advanced cases) constant and very distressing (extreme orthopnea); cough; more or less cyanosis; subcutaneous oedema, etc. In some cases, pain in the region of the heart is complained of, and in one case at least, I have met with well-marked symptoms of angina pectoris. In advanced cases, the radial pulse is unusually small and feeble, often irregular and intermittent; in some cases it is of normal

frequency ; in others slower ; in others again considerably quicker than natural.

In advanced stages of the disease, the impulse of the heart is weak or altogether absent ; the præcordial dulness is usually more or less increased ; the first sound short and feeble, or replaced by mitral or tricuspid systolic murmurs.

Limited fibroid patches do not, as a rule, give rise to any symptoms or physical signs, but to these cases I will again more particularly refer under the head of cardiac aneurisms.

Diagnosis.—The diagnosis of fibroid degeneration of the heart is always somewhat difficult and uncertain. It may be suspected, when there are well-marked symptoms and signs of cardiac weakness, and when the observer is able to satisfy himself that these conditions do not depend upon a chronic valvular lesion, or upon fatty degeneration of the cardiac muscle. In short, in making the diagnosis we *first* determine by the help of the symptoms and physical signs that the heart is weak (*i.e.* chronically weak) ; and *secondly*, by the method of exclusion, we satisfy ourselves that the cause of that weakness is fibroid degeneration, *i.e.* we conclude from the history, general condition of the patient, and physical examination of the heart, that there is no other cause of chronic cardiac weakness present. But to this point I will again more fully refer in treating of the differential diagnosis of fatty heart.

When in addition there is reason to suppose from the history of the case, or from the nature of the physical signs, that the pericardium is adherent, the diagnosis of fibroid degeneration of the heart wall may be more confidently made.

Prognosis.—Chronic myocarditis or fibroid degeneration seriously impairs the functional activity of the part of the heart which is affected by it ; and since the fibroid tissue cannot be removed by treatment, the prognosis is very unfavourable. Sudden death not unfrequently occurs in cases of chronic myocarditis ; in some cases of this description there has been no complaint or suspicion of cardiac derangement

during life ; in some cases, the fatal result it is due to sudden arrest of the cardiac contractions and syncope ; in others, though these are of very rare occurrence, to the rupture of a cardiac aneurism.

Treatment.—Since it is impossible to remove the fibroid patches and to restore the atrophied muscular fibres, the treatment is necessarily palliative. The main indications are to avoid all cardiac strain, and everything likely to cause sudden over-distention of the organ. The patient's life should be emphatically a quiet one, though a certain amount of gentle out-door exercise, sufficient to maintain the general health, but insufficient to cause shortness of breath, or any feeling of cardiac distress, or to produce fatigue, is generally beneficial. The bowels must be carefully regulated, and all straining at stool avoided ; the diet should be light and nutritious, in fact the general measures which have been previously recommended for the treatment of chronic mitral lesions, and into the details of which I need not again enter, are appropriate here. The general health must, at the same time, be kept in the highest possible stage of efficiency. A prolonged course of arsenic, given in gradually increasing doses, in accordance with the capabilities of each individual patient, should be tried ; this is the only drug which in my hands has seemed to produce any real and lasting benefit in chronic myocarditis. Dr Roberts Bartholow speaks hopefully of small doses of the chloride of gold ($\frac{1}{30}$ th of a grain, three times daily). Whenever symptoms and signs of cardiac failure arise they must be met by appropriate remedies, digitalis, alcoholic stimulants, etc. In cases of advanced fibroid degeneration the heart does not respond well to digitalis. In such cases it is often better to rely upon alcoholic and ammoniacal stimulants, spirits of chloroform, etc., rather than to push large doses of digitalis, which, by increasing the arterial blood pressure, necessarily throw increased work upon the damaged organ.

PARTIAL ANEURISMS OF THE HEART.

Definition.—The term aneurism of the heart, which used formerly to be applied to cases in which a cardiac cavity was dilated *as a whole*, is now restricted to those cases in which there is a local bulging or dilatation of some part of the organ. These cases used formerly to be described as *partial* aneurisms of the heart.

Ætiology.—Aneurisms of the heart are either acute or chronic. Both varieties are rare.

Acute aneurisms of the heart may result from anything which causes rapid local softening of a limited portion of the cardiac wall. The resisting power of the affected part of the heart is of course weakened, and it dilates and sometimes ruptures under the internal blood pressure. Acute ulcerative endocarditis, acute localised myocarditis, and acute softening the result of thrombosis of the coronary artery, are the conditions which are most likely to cause acute local dilatations of this description. It has also been thought that abscesses or cysts, which have burst through the endocardium, may form the starting point, as it were, of acute local dilatations. Dr Wickham Legg, in his learned and exhaustive Bradshaw Lecture on Cardiac Aneurisms, to which I would refer the reader for further particulars than can be given here, thinks that these causes are possible, but not yet proved.

Chronic aneurisms of the heart are almost always the result of chronic myocarditis (fibroid degeneration). Fatty degeneration seems to be an occasional, though extremely rare, cause of the condition; Dr Legg cites three cases in which this change was found. In one case, also quoted by the same writer, the aneurism was of traumatic origin, the patient having been stabbed ten years before death in the region of the heart; a scar led from the place of the wound to the apex of the right ventricle; a large bulging of the cardiac wall, which had all the characters of an ordinary aneurism of the left ventricle due to disease, had formed at the seat of the injury.¹

¹ The Bradshaw Lecture on Cardiac Aneurisms. *Medical Times and Gazette*, Aug. 25, 1883, p. 199.

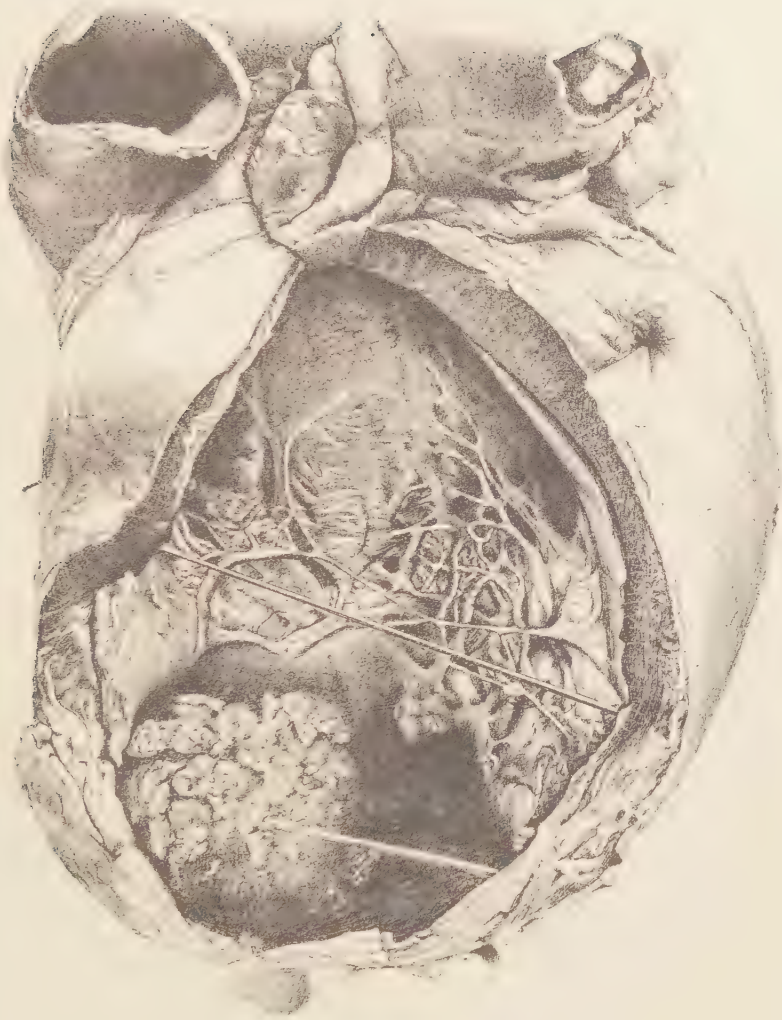


FIG. 242.—*Aneurism of the left ventricle. (Natural size.)*

The left ventricle has been opened in the usual manner; the aneurism, partly filled with laminated fibrine, is seen to be situated at the apex, on the anterior surface of the ventricle, and close to the septum

Copied by Professor Turner's permission from a specimen in the Anatomical Museum of the Edinburgh University

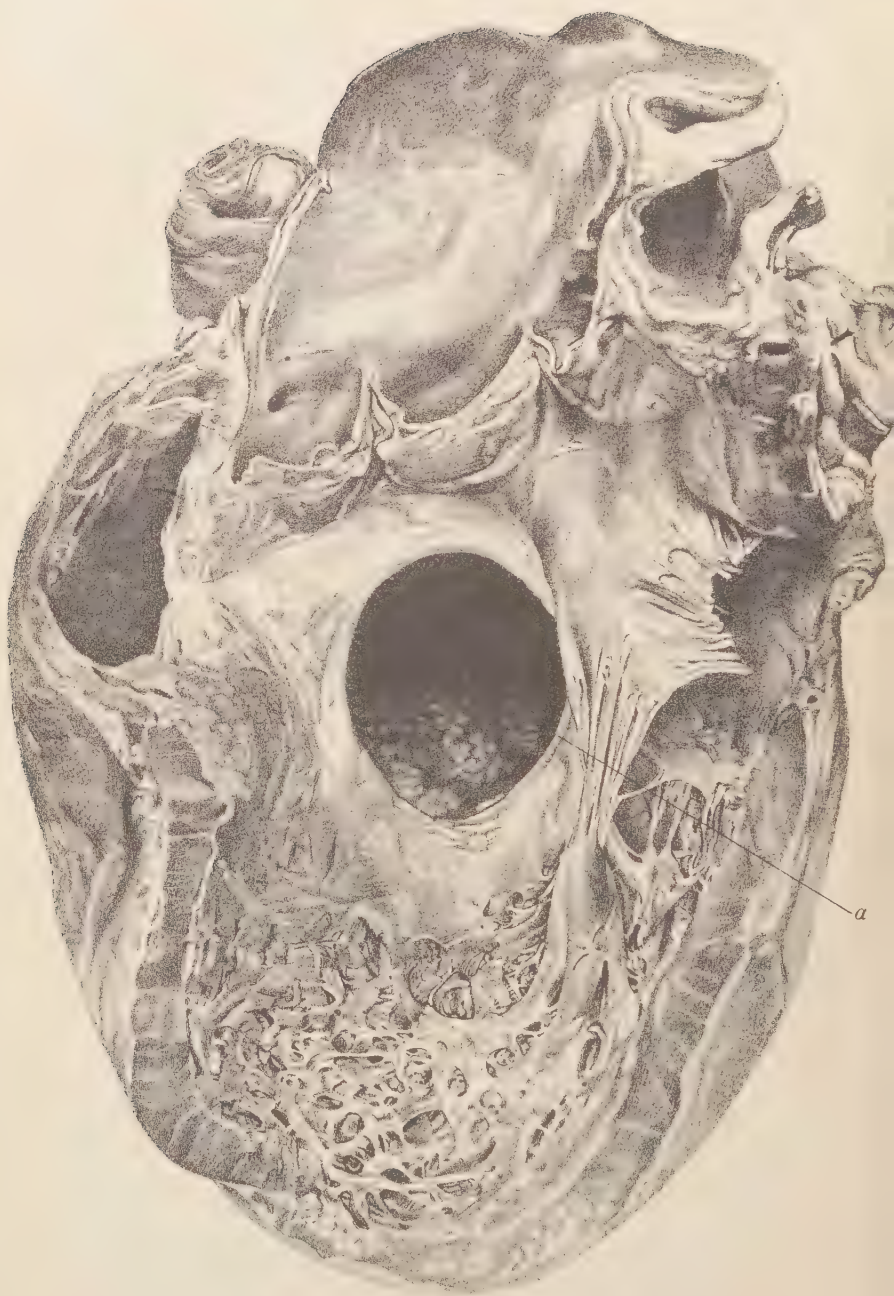


FIG. 243.—*Aneurism of the left ventricle. (Natural size.)*

A large circular opening (*a*) which represents the orifice of the aneurism, is situated in the septum a short distance below the aortic segments.

Copied by Professor Turner's permission from a specimen in the Anatomical Museum of the Edinburgh University.

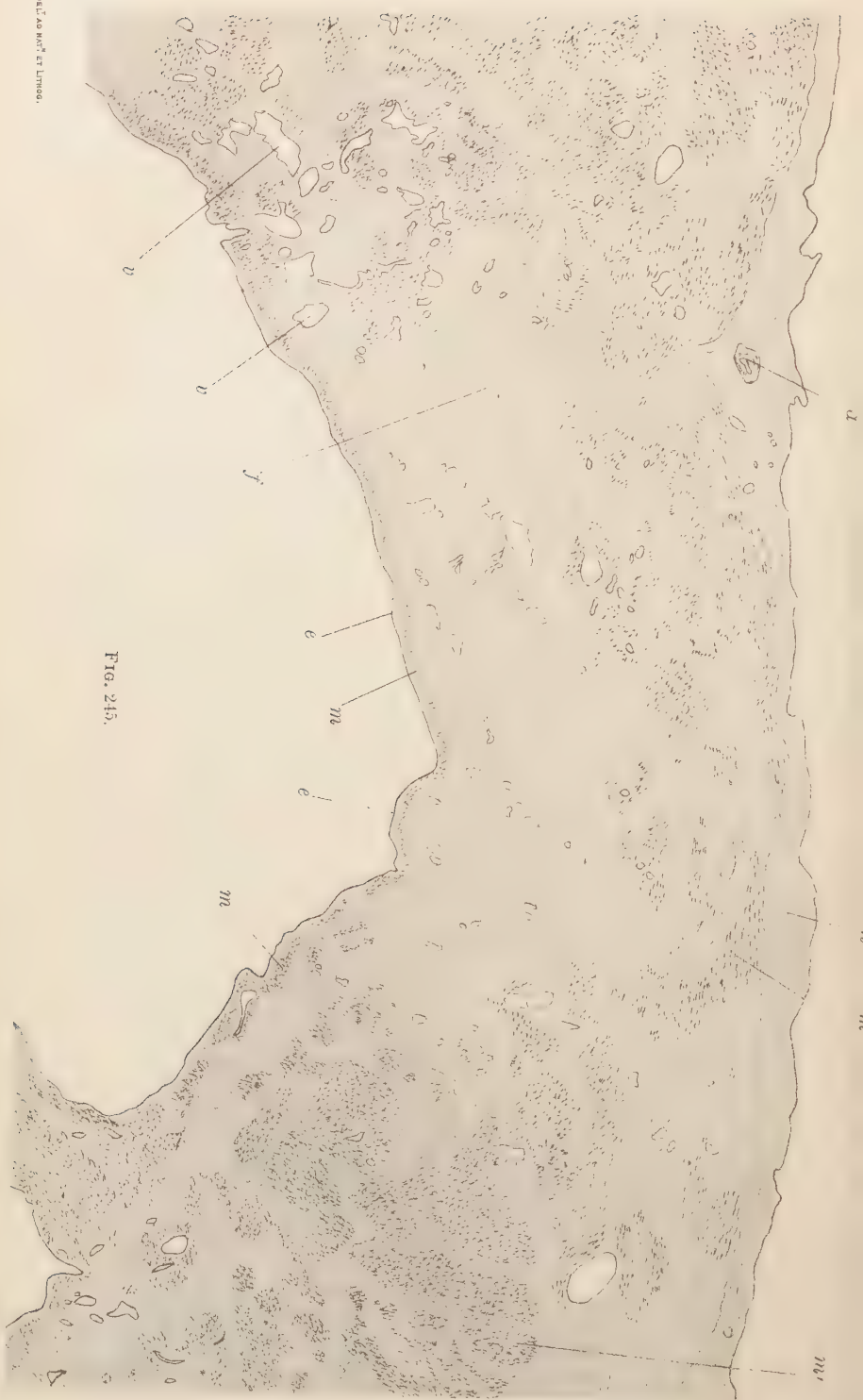


Fig. 245.

Cardiac aneurisms may occur at any age, but are much more common in middle and advanced than in early life.

Pathology.—In the immense majority of cases, both acute and chronic, the aneurismal dilatation is connected with the cavity of the left ventricle. This is, of course, only what we would expect when it is remembered:—(1) that the left ventricle is much more frequently affected by acute ulcerative endocarditis, acute myocarditis, and fibroid degeneration than any of the other cardiac cavities; (2) that the blood pressure in the cavity of the left ventricle is very much greater than in any other part of the organ. The conditions, therefore, for the production of cardiac aneurisms are met with much more frequently, and in a greater degree, in the cavity of the left ventricle than elsewhere.

The aneurism is most frequently situated at the apex of the left ventricle, but it sometimes involves the septum and other parts of the wall of the left ventricle. These appearances are beautifully seen in figs. 242, 243, and 244, which are copied from specimens in the anatomical museum of the Edinburgh University, and which I am able to represent by Professor Turner's kind permission. Dr Legg¹ states that in the cases which he has collected, in fifty-nine cases the aneurism was situated at the apex, and in thirty-one at other parts of the left ventricle. When the aneurism involves the septum, it

¹ Bradshaw Lecture on Cardiac Aneurisms, reported in the *Medical Times and Gazette*, Aug. 25, 1883, p. 199.

Description of Fig. 245.

Section through a commencing aneurism of the left ventricle.

(Magnified about 10 diameters.)

The bulging of the cardiac wall, which constitutes the aneurism, is not seen in the section, which has been flattened out in the process of mounting. The cardiac wall, between *c* and *p* (*i.e.* the wall of the aneurism) is very much thinned; and, at this spot, the muscular fibres have almost entirely disappeared, their place being occupied by fibrous tissue.

e, e, the endocardium at the bottom of the aneurism; *p*, pericardium; *m, m*, a thin layer of muscular fibres adjacent to the endocardium; *m' m'*, muscular fibres in the wall of the heart; *f*, fibrous tissue; *a*, artery in the pericardium, undergoing obliterative endarteritis (more highly magnified in fig. 273; *v, v*, large blood-vessels in the wall of the heart.

projects into the cavity of the right ventricle, in consequence of the fact that the blood pressure in the cavity of the left ventricle is greater than that in the right. Acute aneurisms not unfrequently involve the 'undefended space,' as it is termed,—the highest portion of the septum which is destitute of muscular fibres, and which occupies the angle between the posterior and right segments of the aortic valve.

Cardiac aneurisms vary in size from slight local bulgings up to large tumours, which have been known to attain almost the size of the heart itself. In chronic aneurisms, such as that from which the section represented in figure 245 was made, all three layers of the heart (the endocardium, myocardium, and pericardium) are usually, if not invariably, found in the wall of the sac, but the muscular fibres of the myocardium may be completely replaced by fibrous tissue. In acute aneurisms due to ulcerative endocarditis, the endocardial layer is wanting. Large aneurisms generally contain some laminated coagula.

Symptoms and physical signs.—Aneurisms of the heart are either entirely latent, or attended by the symptoms and signs indicative of chronic myocarditis. Large aneurisms, which are, it is needless to say, of infinite rarity, might give rise to increased dulness on percussion, and pulsation in the region of the heart. Skoda, quoted by Schrötter,¹ observed a bulging of the intercostal spaces overlying the seat of an aneurism.

Diagnosis.—Small aneurisms cannot of course be recognised during life. An aneurism, of sufficient size to produce increased impulse and increased dulness on percussion, would have to be distinguished from simple (general) dilatation of the cardiac cavities and from aneurisms of the aorta. It is most improbable that any of the readers of this work will meet with such a case. The position and outline of the pulsation and dulness, the absence of any of the ordinary causes of cardiac dilatation (such, for example, as stenosis of the mitral orifice), would, I should suppose, be the chief facts suggestive

¹ *Ziemssen's Cyclopædia*, vol. vi. p. 246.

of an aneurism of the heart ; the supposition would, of course, be strengthened if the observer had, before the appearance of the physical signs suggestive of aneurism, made the diagnosis of fibroid degeneration of the heart. These suppositions are, it is needless to say, purely theoretical, no such case having come under my own personal observation.

Prognosis.—The prognosis must, of course, be based upon the general condition of the heart, since it is so rarely possible to suspect the presence of a cardiac aneurism. Cardiac aneurisms sometimes rupture, causing sudden or instantaneous death ; in other cases the patient dies suddenly from syncope. Gradual death may, of course result from the fatty, fibroid, or other cardiac lesions, which happen to be present.

Treatment.—The treatment is that which has been previously recommended for fibroid degeneration. Should the presence of a large cardiac aneurism be suspected, the appropriate treatment for aortic aneurisms should be carried out.

HYPERTROPHY AND DILATATION OF THE HEART.

Hypertrophy and dilatation are closely related, and are very often met with in combination ; hypertrophy, it is true, not unfrequently occurs without dilatation, but dilatation, of the ventricles at least, is almost invariably associated with some hypertrophy. Mechanical causes play a very important part in the production of both conditions ; but the vital state of the organism as a whole, and of the heart in particular, is no less important ; the rapidity, too, with which the mechanical causes to which I shall presently refer more in detail, are established, exerts, in many cases, a very important influence in determining the nature of the result (*i.e.* in determining whether hypertrophy or dilatation will result). An obstruction to the passage of the blood through the minute arteries of the body, which raises the arterial blood pressure and throws an increased strain on the cavity of the left ventricle, is an excellent example of the mechanical causes, which in some cases determine the production of hypertrophy, and in others

of dilatation. When the organism, as a whole, and the heart in particular, is healthy, and when the obstruction to the blood flow is slowly and gradually established, the increased stimulation of the cardiac muscle, and the increased effort demanded of it, result in the production of hypertrophy; when, on the contrary, the vitality of the organism as a whole is below par, when, more especially, the cardiac muscle is degenerated or otherwise diseased, the heart does not respond to the increased stimulation, and instead of becoming hypertrophied and equal to cope with the obstruction, its muscular wall yields under the increased blood pressure, and dilatation is produced. When, again, a considerable obstruction is very rapidly established there may be no time for the production of hypertrophy; in such cases rapid or acute dilatation is produced. The part of the heart, too, which receives the strain and has to put forth the increased effort to overcome the obstruction, is a point of very great importance. The thick left ventricle possesses a much greater degree of reserve force than the thin-walled left auricle, hence its tendency to become hypertrophied rather than dilated; the auricle, on the contrary, tends to dilate rather than to hypertrophy.

Now in a large proportion of the cases, in which mechanical causes of hypertrophy and dilatation are met with in the living man, the organism, as a whole, and the heart in particular, is not quite healthy, the response to the increased stimulus is consequently imperfect, and, while a certain amount of hypertrophy is produced, a certain amount of dilatation is at the same time established. In many cases, too, in which the hypertrophy was in the earlier stages tolerably perfect, and the dilatation at a minimum, the cardiac muscle subsequently degenerates, and the hypertrophy gives place to dilatation. All degrees of combination are therefore met with, from pure hypertrophy without any dilatation on the one hand, to extreme dilatation with hypertrophy at a minimum on the other. If this association be clearly kept in view, we may now conveniently consider hypertrophy and dilatation separately.

HYPERTROPHY OF THE HEART.

Definition.—Increase of the muscular wall of the heart, the result of increase of the muscular tissue. (A hypertrophied heart is heavier, and almost always larger, than a normal heart, but it must be remembered that all large, heavy, and thick-walled hearts are not necessarily hypertrophied. Increased thickness of the cardiac walls may be due to deposits of fat, increase of connective tissue, or the presence of new growths (syphilitic gummata, cancerous, or sarcomatous nodules, etc.); in many cases of this description, to which the term ‘false’ or ‘spurious’ hypertrophy is sometimes applied, the proper muscular elements are in places atrophied, and the thickened heart is actually weaker than normal. Increased size of the heart may also be due to dilatation; but, as I have already explained, some hypertrophy is almost always present in cases of this description).

Varieties.—Three varieties of hypertrophy are usually described, viz. :—

(1) *Simple Hypertrophy.*—In this form the muscular wall of the heart, or rather of the affected cavity of the heart, for it is seldom that all the cavities are implicated, is increased, but the cavity itself is of normal size. The left ventricle in cases of cirrhotic Bright’s disease, often exhibits this form of hypertrophy to perfection. In cases of long-continued bronchitis and emphysema, I have also seen the right ventricle enormously hypertrophied without any dilatation.

(2) *Concentric Hypertrophy.*—This form, in which the muscular wall of the heart is increased and the cavity itself diminished in size (contracted), is probably identical with that previously described (*i.e.* with simple hypertrophy). The apparent diminution in the size of the cavity is, in most cases, if not in all, a *post mortem* phenomenon, and is due to the fact that the heart’s action was arrested in systole, and that the muscular contraction and resulting obliteration of the cardiac cavity continued as the result of *post mortem* rigidity. If, in cases of this description, the heart be steeped for a few hours

in warm water, so as to render its walls flaccid, the cavity which seemed to be obliterated will usually (always in my experience) be found of normal size. It is, in fact, extremely doubtful if concentric hypertrophy ever actually occurs.

(3) *Eccentric Hypertrophy*.—In this form the muscular wall of the heart is increased, while the cavity is at the same time dilated. Eccentric hypertrophy is extremely common, and is met with in its most typical form in the left ventricle of long-continued aortic regurgitation. It is synonymous with the form of dilatation which is technically termed *dilatation with hypertrophy*, in contradistinction to *simple dilatation*.

Ætiology and Pathology.—In some cases the hypertrophy is general, and involves, more or less, all four cavities of the heart; more commonly it is limited to one or two cavities; the ventricles are affected much more frequently than the auricles, and the cavities on the left side of the heart than those on the right; the order of relative frequency with which the four cardiac cavities are affected is therefore :—

Left ventricle.

Right ventricle.

Left auricle.

Right auricle.

It is a disputed point, whether in cases of cardiac hypertrophy there is an actual increase in the number of the muscular elements, or whether the original muscular fibres are simply increased in size. The question is one which is not of any great practical importance, I need not, therefore, enter into details, suffice it, however, to say, that both alterations probably occur.

Speaking generally, it may be said that the cause of hypertrophy of the heart is increased stimulation and over-work. In the great majority of cases, the primary cause is mechanical, such, for example, as obstruction to the blood-flow either inside or outside the organ. Hypertrophy seems also sometimes to be caused by long-continued

over-activity, the result of neurotic or other conditions, independently of obstruction to the blood-current or other mechanical causes.

It is extremely important to remember that for the production of satisfactory hypertrophy the cardiac muscle must be healthy, and the conditions for its nutrition satisfactory. When, for instance, the muscular fibres are degenerated, the heart may be incapable of becoming hypertrophied, as in the case of cirrhosis of the kidney and fibroid degeneration of the heart, which I have previously described. So again, when the arterial blood supply to the cardiac walls is insufficient in consequence of disease of the coronary arteries, or when the waste products of combustion are retained in the heart in consequence of venous congestion, the hypertrophy is imperfect, or, if it should have been previously established, gives place to dilatation in consequence of fibroid or fatty changes in the thickened, and, at the same time, imperfectly nourished muscular walls of the organ.

The chief causes of hypertrophy are therefore as follows:—

A. *Extrinsic causes.*

1. *Long-continued simple over-activity or functional excitement.*—Under this head are included those cases of hypertrophy of the heart which result—(a) from physiological over-activity, such as is necessitated by violent muscular effort; (b) from long-continued neurotic palpitation due to any cause, such as exophthalmic goitre, sexual excesses or irregularities, etc. The occurrence of cases of this description, which are sometimes called cases of ‘*idiopathic*’ hypertrophy, is doubted by some observers; but while allowing that they are seldom seen on the *post mortem* table, for the conditions with which they are associated are rarely fatal, I am perfectly convinced, from the clinical examination of living patients, that they do actually occur. In these cases the hypertrophy is, as a rule, *general*, i.e. it involves both the left and the right hearts.

2. *Obstruction to the blood-flow outside the heart.*—This is an extremely common cause of hypertrophy both of the left and of the right ventricles. Any obstruction, for example, to

the passage of the blood through the systemic arterial system will, if long continued, produce hypertrophy of the left ventricle; while, hypertrophy of the right ventricle follows any long-continued obstruction in the pulmonary circuit. The different pathological conditions, which produce hypertrophy of the left and right ventricles, and which are included under this head, will be presently detailed. In cases of this description the hypertrophy is, in many cases, *pure*, *i.e.* unassociated with dilatation.

3. *Obstruction to the blood-flow in the heart itself.*—This, too, is one of the most common causes of cardiac hypertrophy. The cavity, which is situated immediately behind the obstruction, becomes first hypertrophied; in the case of aortic stenosis, for example, the left ventricle is first affected, in the case of mitral stenosis the left auricle, and so on.

4. *Increased distention of a cardiac cavity.*—When, for example, the left ventricle receives an unusually large quantity of blood, as it does in cases of aortic and mitral regurgitation, its muscular wall is unduly stimulated, and an increased effort is required to empty it; hypertrophy is in consequence produced. In cases of this description the hypertrophy is always combined with dilatation.

5. *An impediment to the free contraction of the heart, such as is present in some cases of adherent pericardium.*—Cases of adherent pericardium are not unfrequently met with, in which the heart is hypertrophied; in many of these cases, the hypertrophy is due to associated valvular disease; but in some, it is, I think, in part at least, due to the impediment to the free action of the organ, which is caused by the pericardial adhesions. The heart is most likely to become hypertrophied when the pericardium is adherent to the chest-wall on the one hand, and to the exterior of the heart on the other. When extensive pericardial adhesions of this description are present, there is often at the same time extensive fibroid degeneration of the myocardium. In such cases the cardiac muscle may be so extensively degenerated as to be incapable of responding to the extra exertion demanded of it. In fact, in many cases of adherent pericardium

the muscular tissue of the heart is more or less atrophied ; and it is in cases of this description, in which the walls of the heart are often much thickened, that a careless observer is likely to mistake the 'spurious' or 'false' hypertrophy for the 'true' form of the condition.

Let us now consider the hypertrophies of the left and right hearts, and of the individual cavities in detail. This division of the subject has many practical advantages, and is amply justified by the results of clinical and *post mortem* observation. It must, however, be remembered that hypertrophy of either heart is very generally associated with some hypertrophy of the other. The affection of both hearts is sometimes due to the fact, that the original cause of the hypertrophy, although situated in one heart, ultimately throws an increased strain on the other. Aortic regurgitation, for example, first causes hypertrophy of the left ventricle, but ultimately causes such engorgement of the pulmonary circulation, that hypertrophy of the right ventricle is produced. So, too, great obstruction originating in the lungs, as in cases of long-continued bronchitis and emphysema, first produces hypertrophy of the right heart, but ultimately may cause some hypertrophy of the left ventricle, in consequence of the difficulty which the arteries have in emptying themselves into the greatly engorged and now distended veins. The subcutaneous dropsy which is usually present, also mechanically impedes the systemic peripheral circulation. Further, the venous condition of the blood adds to the difficulties of the arterial circulation, *firstly*, by causing increased capillary resistance, for venous blood passes with difficulty through the capillary vessels, and *secondly*, by stimulating the vaso-motor centre, and producing contraction of the minute arteries.

Probably, too, it is impossible to have much hypertrophy of one ventricle without some hypertrophy of the other, in consequence of the fact that the two ventricles are in the habit of acting harmoniously together, and that the tissues of the one are to some extent, at least, continuous with the tissues of the other. In a case, which recently came under my observation in the *post-mortem* theatre, in which the right

heart was enormously hypertrophied in consequence of long-continued bronchitis and emphysema, it seemed difficult to account for the great hypertrophy of the left ventricle, which was also present, on any other supposition.

HYPERTROPHY OF THE LEFT VENTRICLE.

Ætiology.—The left ventricle is the part of the heart which is most prone to hypertrophy. The chief clinical and pathological conditions which produce hypertrophy of the left ventricle are as follows:—

A. *Intrinsic causes*—*i.e.* conditions within the left heart.¹

(1) *Aortic Stenosis.*—In this condition, the hypertrophy is as a rule ‘simple,’ *i.e.* there is little or no associated dilatation. The hypertrophy results from the increased effort which is required to force the blood through the narrowed aortic orifice.

(2) *Aortic Regurgitation.*—Here the hypertrophy is of the ‘*eccentric*’ variety; the associated dilatation is often considerable, and the hypertrophy great; in fact it is in cases of aortic regurgitation that the largest and heaviest hearts occur. It was stated recently in one of the medical journals (I have unfortunately lost the reference), that a heart was shown to the Medical Society of New York which weighed no less than 57 ounces. Dr Bristowe has recorded another which weighed 46½ ounces, and I myself have met with an example (the case detailed on page 501) in which the weight of the heart was 45 ounces. When the aortic valve is incompetent, blood is poured into the cavity of the left ventricle during the ventricular diastole from two sources, *viz.*, from the aorta on the one side, and from the left auricle on the other; the forcible distention of the cavity, the walls of which are flaccid, produces, from the first, some dilatation; the muscular wall of the cavity is more forcibly and more frequently stimulated than in health; and there is at the same time an unusually large quantity of blood to be discharged. These conditions

¹ It will be observed that I use the terms ‘extrinsic’ and ‘intrinsic’ as applied to the left heart, and not to the heart as a whole.

(excessive stimulation and the increased effort required to expel the abnormally large contents) necessarily lead to hypertrophy.

(3) *Mitral incompetence*.—In free mitral regurgitation the left ventricle is almost always increased in thickness; the hypertrophy being of the ‘*eccentric*’ variety, *i.e.* associated with some dilatation. (In some cases the hypertrophy is of the ‘*spurious*’ or ‘*false*’ form, but in most cases true hypertrophy is present). Considerable difference of opinion has existed as to the manner in which the hypertrophy is produced in cases of this description. (*a*) The chief cause is, I believe, excessive stimulation of the muscular wall, and the increased effort which is required to expel the abnormally large quantity of blood which the cavity contains. In cases of free mitral regurgitation the left auricle and the whole pulmonary circuit are greatly distended during the ventricular systole; as soon as the contraction of the left ventricle ceases, the mitral valve yields before blood pressure, and an unusually large quantity of blood passes with great force into the left ventricle, the walls of which are in a flaccid and relaxed condition. Some dilatation of the left ventricle is consequently produced, its muscular wall is more forcibly and frequently stimulated than in health, and an increased effort is required to expel its abnormally large contents; hence the production of hypertrophy. But whilst this is the main cause, the hypertrophy is probably in part due to the fact (*b*) that the left ventricle shares in the hypertrophy of the right heart, which is present (this point has been previously referred to on page 585); possibly, too, in the later stages of mitral disease, to (*c*) the difficulty which is offered to the passage of the blood from the systemic arterial system into the engorged veins. (This cause of hypertrophy has also been explained. See page 585.) The hypertrophy of the left ventricle, which is met with in some cases of mitral stenosis (in which the left ventricle is, as a rule, of normal size, or smaller than normal) is probably also produced in the manner described under the two latter heads (*b* and *c*).

(4) *Pericarditis, with extensive adhesions*.—There is seldom

much true hypertrophy of the left ventricle in such cases. (The manner in which pericardial adhesions may produce cardiac hypertrophy has already been described. See page 584.)

B. *Extrinsic Causes*—(*i.e.* causes outside the left heart.)

1. *Bright's Disease of the Kidney*.—Hypertrophy of the left ventricle is seen in all forms of Bright's disease (the waxy or lardaceous, when uncombined with cirrhosis, excepted), but reaches its highest degree in the cirrhotic variety. In cases of this description, there is little or no dilatation, *i.e.* the hypertrophy is of the '*simple*'¹ variety. Various theories have been advanced to account for the hypertrophy of the left ventricle which is so constantly present in cirrhotic Bright's disease. All observers are agreed *firstly*, that the cause of the hypertrophy is situated outside the heart, *i.e.* in some part of the arterial or capillary systems; *secondly*, that in consequence of this obstruction in the arterial or capillary system of vessels, the arterial blood pressure is increased; and *thirdly*, that the left ventricle has consequently more work to do, and therefore becomes hypertrophied. But opinions differ as to the exact manner in which the obstruction is produced.

Traube believed that the increased arterial tension resulted from the difficulty which the blood meets with in passing through the cirrhotosed kidneys, a part of the capillary system of the kidneys being cut off, as it were, by the fibroid lesion.

Sir William Gull and *Dr Sutton* suppose that the cirrhosis of the kidney is only part and parcel of a vascular lesion, arterio-capillary sclerosis, as they term it, which is distributed throughout the whole body; and that the increased arterial tension is due to the difficulty which the blood meets with in passing through the minute arteries and capillaries, the calibre of which is narrowed by the fibro-hyaloid change which they claim to have demonstrated.

¹ I do not refer to the 'concentric' form, which is, I believe, an unreal form; it is, however, in cases of 'contracted' kidney that this supposed concentric hypertrophy is most frequently observed.

Dr George Johnson thinks that in consequence of the kidney affection, excrementitious products, urea and its compounds, are retained in the blood ; that the muscular coats of the minute arteries are irritated and kept in a spasmodic state of contraction, and become hypertrophied ; and that in consequence of these arterial changes the calibre of the minute arteries throughout the body is narrowed, the arterial tension increased, and an increased strain thrown upon the left ventricle.

Dr Broadbent is of opinion that, while contraction of the arterioles may give rise to very high arterial tension, and no doubt often co-operates with capillary resistance in its production, the seat of the obstruction from which high pressure within the arterial system usually arises, is situated in the capillary system of vessels.¹

Space does not permit me to discuss in detail the various facts and arguments which have been brought forward in support of these different views. Suffice it to say, that I agree with those who think *firstly*, that the primary cause is the kidney lesion ; *secondly*, that in consequence of the kidney lesion the blood becomes loaded with excrementitious matters, and that it is owing to the presence of these impurities in the blood that the increased arterial tension is produced. It is much more difficult to decide the *third* step in the process, and to determine the exact manner in which the increased arterial tension is brought about, *i.e.* whether it is due to increased capillary resistance or to obstruction in the minute arterioles ; probably both views are correct. So far as I can weigh the facts, I am disposed to think that the chief cause of the obstruction must be in the minute arteries. At the same time I am quite willing to allow that impure blood probably passes through the capillaries with greater difficulty than healthy blood. I am disposed, therefore, to think that the increased arterial tension of cirrhotic Bright's disease is due, partly to increased capillary resistance, partly and chiefly to increased contraction of the minute arteries. The over-contraction of the arteries, or arterial spasm, as it has been

¹ *British Medical Journal*, Aug. 25, 1883, p. 357.

termed, is possibly produced by the direct irritation of urea and its products, possibly, as Ludwig has supposed, by the action of the retained urinary products on the vaso-motor centre. Perhaps, too, it may be partly caused, as Israel and Grawitz (quoted by Dr Saundby in the debate on increased arterial tension at the Liverpool Meeting of the British Medical Association¹), by the direct action of the retained urinary products on the cardiac ganglia or muscle of the heart. (These observers have shown that high arterial tension may be produced by feeding animals on urea. It is interesting in this connection to remember the close relationship which, as Gaskell and others have shown, the cardiac muscle bears to unstriated muscular fibre, and the fact that some substances, *e.g.* digitalis, which stimulate the muscular fibre of the heart, also act upon the muscular coats of the arteries. It is highly probable, therefore, that if urea directly stimulates the cardiac muscular fibre, it also directly stimulates the muscular coat of the minute arteries—an additional argument to the others which can, I think, be legitimately advanced to show that the contraction of the arterioles exercises a very important, I would say, the most important part, in the production of the high arterial tension of Bright's disease.)

2. *Atheroma*.—All observers agree that a rigid inelastic condition of the arteries causes a decided obstruction to the blood current, and throws an increased strain on the left ventricle, which tends, therefore, to become hypertrophied. In some cases of this description, the cardiac muscle is fatty or otherwise degenerated, and incapable of much hypertrophy; dilatation then of course results.

3. *Constriction of the aorta or large blood-vessels*.—The thoracic aorta is sometimes constricted as the result of congenital malformation (the constriction is usually situated just beyond the ductus arteriosus Botalli); and in these cases the left ventricle has been found hypertrophied. Spinal curvatures and intra-thoracic tumours, which press upon and constrict the aorta or its large branches as they leave the narrow upper outlet of the thoracic cavity, may also

¹ Reported in the *British Medical Journal* for Aug. 25. 1883, p. 361.

act as mechanical causes of hypertrophy of the left ventricle. Obstruction of the abdominal aorta may also have the same effect. The influence which aneurismal dilatations of the aorta, more especially saccular aneurisms, have, in causing hypertrophy of the left ventricle, is doubtful; theoretically we would expect hypertrophy to be produced, more particularly in cases of general dilatation; in some cases it certainly does occur, but not in all. It is frequently absent when the aneurism is saccular.

4. *Pregnancy*.—The hypertrophy due to this cause is so well known as to need no comment.

5. *Chlorosis and progressive pernicious anæmia*.—In these conditions, arterial tension is usually increased, partly, I believe, in consequence of the difficulty with which anæmic blood passes through the systemic capillaries, partly because of the contracted condition of the minute arteries, produced by stimulation of the vaso-motor centre. (Physiologists have shown that the vaso-motor centre is stimulated not so much by an excess of carbonic acid in the blood as by a deficiency of oxygen.)

6. *Pulmonary lesions* (such as emphysema and cirrhosis) *and lesions of the right heart*.—In long-continued cases of this description the left ventricle may ultimately become hypertrophied. The exact mode of causation has been previously described. (See page 585.)

Symptoms and consequences of hypertrophy of the left ventricle.—In considering the symptoms and consequences of hypertrophy of the left ventricle, it must always be remembered, that in the great majority of cases the condition is secondary, and that it is for the most part a salutary change.

The hypertrophy itself may produce no discomfort to the patient; and even when it does produce symptoms, they are almost always secondary and subordinate to those which result from the primary lesion. The symptoms, which may result from the hypertrophy itself, are:—consciousness of the action of the heart (uneasiness, a feeling of distention in the

præcordial region, intermittent action, and, above all, palpitation); shortness of breath; symptoms due to over-distention of the vessels, such as throbbing of the arteries, fulness of the heart, headache, flushings, noises in the ears, flashes before the eyes, etc. Palpitation is very common in the advanced stages of hypertrophy, and is suggestive of commencing dilatation; it indicates, as a rule, that the enlarged heart is, in consequence of degenerative (fibroid or fatty) changes, unable to cope with this obstacle in front; or, to use the expressive words of Dr Fothergill, 'palpitation is the outward visible sign of internal incompetence.'¹

The injurious results which may follow hypertrophy of the left ventricle are atheroma and aneurismal, or simple dilatation of the arteries, and arterial rupture. These results do not, of course, always occur; they are only likely to be produced in those cases in which an excessive quantity of blood is propelled by the hypertrophied left heart into the arterial system. They are most likely to occur in atheroma, in kidney disease, and in those cases of aortic incompetence in which there is no obstruction at the aortic orifice. In cases of this description, the force of the hypertrophied left ventricle is, as it were, expended on the arterial system, and if the arteries are degenerated, aneurism and rupture may of course arise. We know, too, that sudden and forcible distention of the arteries, when long continued, is a fertile source of atheroma. Long continued and forcible over-distention of the arterial system (such as is met with in some cases of cardiac hypertrophy), may, therefore, lead to the formation of aneurisms and to arterial ruptures, *firstly*, by causing arterial degeneration (*i.e.* atheroma), and *secondly*, by causing the degenerated and weakened vessel to dilate or give way.

In those cases of hypertrophy, on the other hand, in which the force of the left ventricle is not expended on the arterial system, as for example, in stenosis of the aortic orifice and incompetence of the mitral, these injurious consequences do not occur. In aortic stenosis, the hypertrophy of the left ventricle is altogether salutary; in mitral incompetence, it

¹ *Diseases of the Heart*, p. 127.

is not altogether beneficial, for the greater the force with which the regurgitant current is driven backwards through the mitral orifice, the greater will be the dilatation of the left auricle and the engorgement of the parts behind it, other things, more particularly the extent of the mitral leak, being equal.

Physical signs.—The physical signs which are met with in cases of hypertrophy of the left ventricle vary with the nature of the primary lesion on which the hypertrophy depends. When the hypertrophy is due to mitral regurgitation or aortic incompetence, for example, the *primary* physical signs, as I term them, of those lesions will be present, in addition to those which result from the hypertrophy.

The physical signs characteristic of hypertrophy of the left ventricle *per se* (i.e. the primary physical signs of hypertrophy, as they may be termed) are the physical results of an enlarged and forcibly contracting left heart. It must, however, be remembered that these physical signs are not invariably present, for if the enlarged and forcibly acting left ventricle is extensively covered by lung, as it is, for example in pulmonary emphysema, the physical indications may not be apparent.

The *apex beat* is displaced downwards and outwards, and may be situated in the sixth, seventh, or even the eighth interspace, one, two, or even three inches outside the line of the nipple. (See fig. 246.) (When the apex is much displaced the heart is usually dilated as well as hypertrophied.)

The *impulse* of the heart is more forcible than in health and in many cases the front wall of the chest is forcibly raised up, as it were, *en masse*. The contraction of the organ, and therefore the impulse, are often more deliberate than in health. A double impulse or shock is sometimes perceptible, the first and chief impulse corresponding to the systole of the ventricle, the second and weaker impulse to the closure of the aortic segments and to the cardiac rebound.

The præcordial region is, in some cases (more especially in children and young persons in whom the chest-wall is soft and yielding), more prominent than in health.

The *areas of cardiac dulness*, both superficial and deep are increased, and the percussion resistance is greater than in health. In hypertrophy of the left ventricle the cardiac dulness is increased in the vertical, and to some extent also in the transverse directions, for the hypertrophy is usually, as I have previously pointed out, associated with some dilatation. (See fig. 246.)

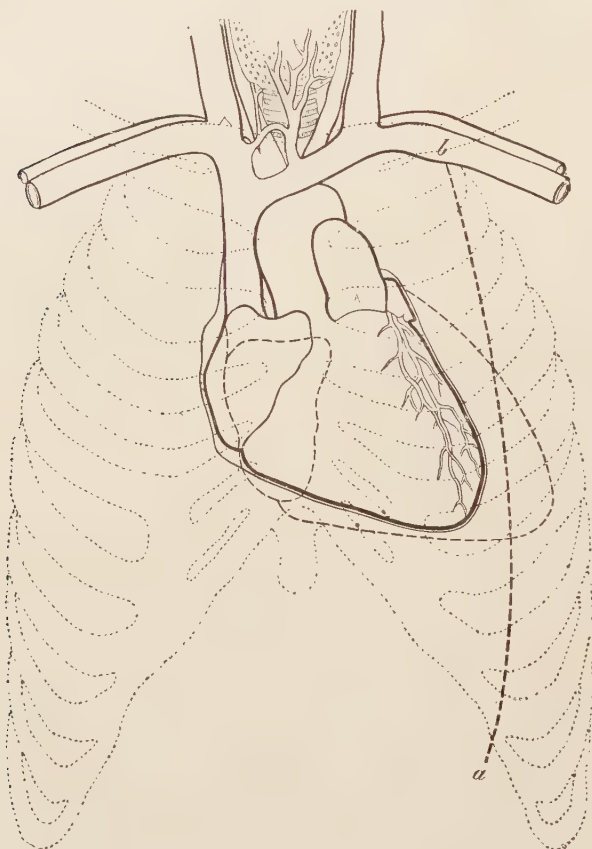


FIG. 246.—*Hypertrophy of the left ventricle, showing the altered position of the apex.*
—(After von Dusch.)

The continuous line represents the normal heart; the dotted line the hypertrophied left ventricle. The apex of the left ventricle is outside the dotted line *a b*, which is drawn through the left nipple.

The *character of the heart sounds* depends more particularly upon the nature of the primary lesion. When the hypertrophy depends upon extra-cardiac conditions, *i.e.* when there is no valvular disease, and when the heart is (excepting the hypertrophy of the left ventricle) otherwise healthy, the impulse of the hypertrophied heart can sometimes be heard against the chest-wall as a jar or click; the first sound is usually more prolonged, more muffled and duller than in health, and not unfrequently reduplicated; when the hypertrophy is combined with dilatation, the first sound is, on the contrary, louder and shorter than natural; the aortic second sound is, as a rule, loud and accentuated. When the hypertrophy depends upon intra-cardiac causes, such as aortic or mitral disease, the sounds are, of course, modified in accordance with the nature of the valvular lesion.

The *character of the cardiographic tracing*.—The systolic portion of the tracing is very prominent, the summit being broad and sustained, and the eminence, *f*, well marked. (See figs. 247 and 248.)

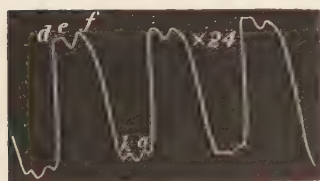


FIG. 247.

FIG. 247.—*Cardiographic tracing in hypertrophy of the left ventricle.* (After Galabin.)

‘Emily L., æt. 8. Loud systolic murmur at the apex, preceded by a very faint rumbling sound. A presystolic murmur has been heard previously. Heart much hypertrophied, P. 96.’ (*Guy’s Hospital Reports*, 1875, p. 313.)

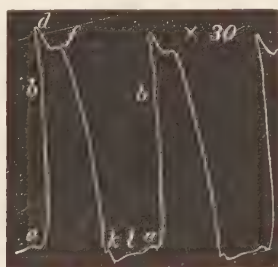


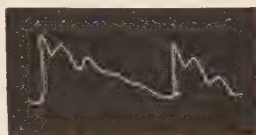
FIG. 248.

FIG. 248.—*Cardiographic tracing in hypertrophy of the left ventricle.*—(After Galabin.)

‘Thomas G., æt. 56. Chronic Bright’s disease with atheromatous arteries. The cardiac impulse was very powerful, but no murmur was heard, P. 63.’—(*Guy’s Hospital Reports*, 1875, p. 312.)

The *character of the pulse* also varies in accordance with the cause of the hypertrophy. When the hypertrophy is due to extra-cardiac conditions, such as cirrhotic Bright's disease, the pulse is firm, strong, and sustained; sometimes full and large, but more frequently of normal volume or contracted. When the hypertrophy depends upon intra-cardiac conditions, the character of the pulse is variously modified in accordance with the special nature of the valvular lesion.¹ The heart's action, and therefore the pulse, is, as a rule, regular; and in this respect there is a striking difference between cases of typical hypertrophy and cases of typical dilatation; in the latter condition marked irregularity is frequently present.

The character of the sphygmographic tracing depends upon the condition of the valvular apparatus of the heart and to a considerable extent upon the state of the peripheral



Pressure 5 ozs.

FIG. 249.—*Sphygmographic tracing from a case of Chronic Bright's disease, with hypertrophy of the left ventricle.*

The pulse is one of high tension: the tidal wave is strongly marked.

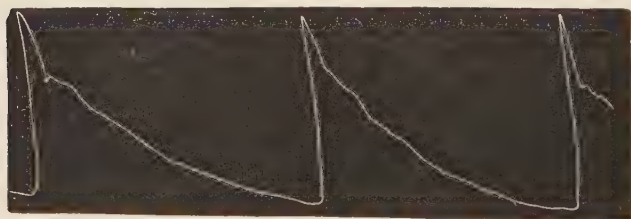
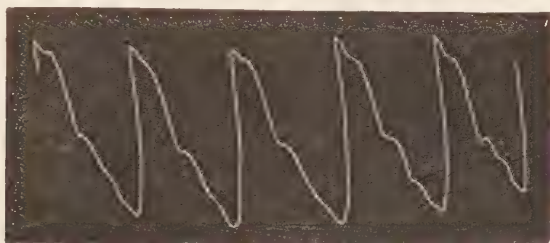


FIG. 250.—*Sphygmographic tracing from a case of mitral disease, after the administration of digitalis.*

The heart was much hypertrophied; the pulse was intermittent, each of the pulse curves shown in the figure occupies the time of two ordinary cardiac revolutions.

¹ The special characters of the pulse in aortic and mitral lesions are described on pages 444, 492, 513, and 525.

vessels (whether firmly contracted, atheromatous, etc.); the tidal wave is usually very prominent, and the systolic portion of the tracing more sustained than normal; in those cases in which the peripheral vessels are not very tightly constricted the up-stroke is taller than normal. The tracings shown in figs. 249, 250, and 251, illustrate some of these points.



Pressure $3\frac{3}{4}$ oz.

FIG. 251.—Pulse tracing in a case of hypertrophy of the left ventricle (probably depending upon cirrhosis of the kidney).

HYPERTROPHY OF THE RIGHT VENTRICLE.

Ætiology.—Hypertrophy of the right ventricle is much more frequently due to extrinsic than to intrinsic causes, *i.e.* to conditions without, rather than within, the right heart.¹ More or less dilatation is almost always associated with hypertrophy of the right heart, and in the majority of cases the dilatation is in excess of the hypertrophy. In this respect, therefore, hypertrophy of the right ventricle differs somewhat from hypertrophy of the left. Cases are, however, occasionally met with in which the right ventricle is greatly hypertrophied, and in which there is little or no dilatation. (I have before me the heart of a patient who suffered for many years—from youth upwards,—from chronic bronchitis and emphysema; the right ventricle is enormously hypertrophied, but the cavity is not dilated.) Cases of this description are, however, rare, and probably only occur in young subjects in whom the recuperative powers, which favour the production of hypertrophy, are great, and in whom the cardiac muscle is perfectly healthy.

¹ The reader must remember that the terms 'extrinsic' and 'intrinsic' are here applied not to the heart *as a whole*, but to its right half only.

The conditions which produce hypertrophy of the right ventricle are as follows :—

A. Extrinsic Causes.

1. *Mitral lesions.* (Both stenosis and incompetence.)—The right ventricle is generally found (after death) to be considerably dilated as well as hypertrophied. In the earlier stages (but probably only in young subjects and in cases in which the cardiac muscle is not degenerated), the hypertrophy may for a time be simple, *i.e.* unattended by any dilatation.

2. *Primary lung disease.*—Emphysema, cirrhosis, and all lung diseases, which interfere with the passage of the blood from the right to the left heart, throw an increased strain on the right ventricle, and tend to produce hypertrophy. In exceptional cases the hypertrophy is, as I have just pointed out, attended with little or no dilatation, *i.e.* is simple; but in the large majority, dilatation is not only present, but is in excess of hypertrophy.

3. *Narrowing of the pulmonary artery in consequence of external pressure.*—This is an extremely rare cause of hypertrophy of the right ventricle, but it does occasionally occur.

B. Intrinsic Causes.

1. *Pulmonary stenosis.*—In cases of congenital pulmonary stenosis, the right ventricle is not always hypertrophied, for the foramen ovale frequently remains patent, and by allowing the blood to pass directly from the right to the left auricle, takes off the strain from the right heart. In those cases of pulmonary stenosis, in which the blood has to pass through the lungs in order to get to the left heart, the right ventricle does become hypertrophied.

2. *Pulmonary incompetence.*—Incompetence at the pulmonary orifice is so infinitely rare, that, for practical purposes, it may be left out of account. It would produce hypertrophy and dilatation of the right ventricle, just as incompetence of the aortic valve produces hypertrophy and dilatation of the left.

3. *Tricuspid incompetence.*—Primary tricuspid incompetence is rare. It would probably be attended by some hypertrophy

of the right ventricle. Secondary tricuspid incompetence (the result of mitral lesions or primary lung disease) occurs after the right heart has become dilated, and can hardly, therefore, be expected to produce much or any hypertrophy of the right ventricle. The very fact that tricuspid incompetence is established, shows that the right heart is failing, and unequal to meet the demands upon it, *i.e.* is unable to become hypertrophied.

4. *Pericarditis, with adhesions.*—The effect of pericardial adhesions in producing hypertrophy of the left ventricle has already been described. The same remarks apply to the right heart, which is probably more apt to become hypertrophied and dilated in consequence of pericardial adhesions, than the left.

Symptoms and consequences of hypertrophy of the right ventricle.—In the great majority of cases, hypertrophy of the right ventricle is secondary to some obstruction in front. The symptoms with which it is associated are usually, therefore, those of mitral or lung disease, shortness of breath being one of the most prominent. The hypertrophy itself is conservative and salutary. Hypertrophy of the right ventricle may be attended with a feeling of tightness, uneasiness, beating, etc., in the pit of the epigastrium, but sensations of this description are usually associated with dilatation rather than with simple hypertrophy. The condition is not, in fact, attended by any characteristic symptoms. When dilatation is combined with hypertrophy, symptoms of engorgement of the systemic veins and of embarrassed pulmonary circulation are usually present.¹ The more the dilatation exceeds the hypertrophy the greater will be the venous engorgement, the greater, therefore, the symptoms, and *vice versâ*.

The pulmonary symptoms are in part due to the defective blood supply to the lungs, which results from the dilated condition of the right heart, but chiefly to the primary lesion—mitral disease, or bronchitis and emphysema, for example.

¹ Should the tricuspid give way, the usual physical signs of that condition (systolic tricuspid murmur, jugular pulsation, etc.) will, of course, be present.

Though hypertrophy of the right ventricle is a compensatory condition (for it enables the right heart to cope with an obstacle in front) it is not an unmixed good, for the increased blood pressure, which is present in the pulmonary circuit (the result partly of the obstruction in front and partly of the increased pressure from behind) is apt to lead to rupture of the pulmonary capillaries, and to produce hæmoptysis, pulmonary apoplexy, atheroma of the pulmonary artery, etc.

The physical signs of hypertrophy of the right ventricle are those of an enlarged and powerfully acting right heart. The left apex beat is, as a rule, more diffused and displaced a little to the left (but not *downwards* and to the left as it is in hypertrophy of the left ventricle), pulsation is often visible in the pit of the epigastrium, or between the left apex beat and the lower end of the sternum. On palpation the pulsation of the right heart is felt to be stronger than in health. The cardiac dulness is increased, more especially in the transverse direction and to the right of the sternum. (See fig. 252.) The first sound over the right ventricle is, as a rule, louder and sharper than in health (hypertrophy of the right heart is almost always associated with a considerable degree of dilatation); the second pulmonary sound is accentuated, and sometimes reduplicated. The radial pulse is, in most cases, small and weak (in consequence of the mitral lesion or lung disease with which the hypertrophy of the right ventricle is associated), and it never presents the firm hard sustained character which it has in many cases of hypertrophy of the left ventricle. The venous circulation is often congested. (Congestion of the systemic venous circulation is a sign of failure of the right heart rather than of increased strength, and is indicative of dilatation rather than hypertrophy; it must, however, be remembered that some dilatation is almost always combined with the hypertrophy, and, as a matter of fact, some venous engorgement is not uncommon in cases in which signs of hypertrophy of the right heart are well marked.)

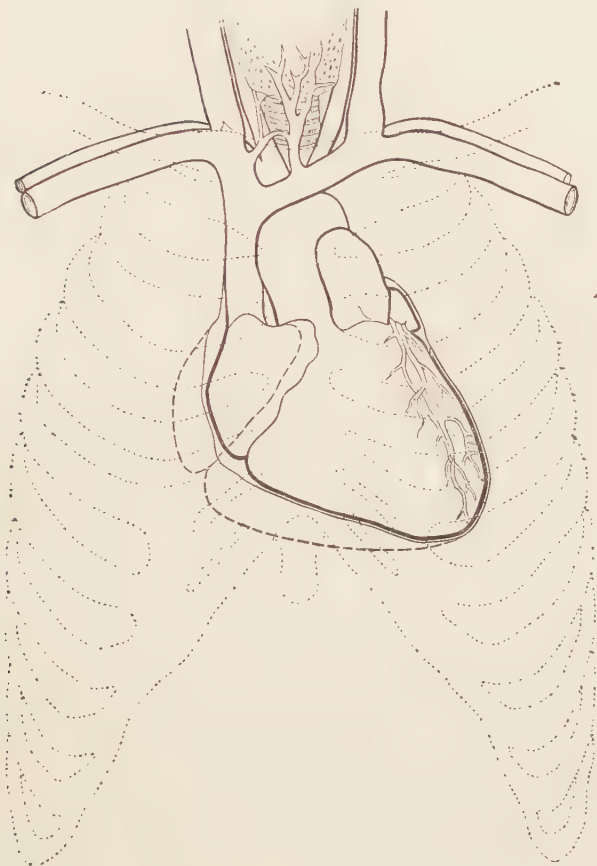


FIG. 252.—*Hypertrophy of the right ventricle.*—(After von Dusch.)

The normal outline of the heart is represented by a continuous line; the hypertrophied right heart by a dotted line.

HYPERTROPHY OF THE AURICLES.

The auricles are much more prone to become dilated than hypertrophied.

Hypertrophy of the Left Auricle is more frequently met with than hypertrophy of the right; it results from mitral

stenosis, and reaches its highest degree of development, though that is never great, in young persons, and in those cases in which the cardiac muscle is sound and capable of considerable hypertrophy. Hypertrophy of the left auricle is, as we have previously seen, one means by which an obstruction at the mitral orifice is compensated, and is, therefore, beneficial. It also occurs in some cases of mitral regurgitation. Hypertrophy of the left auricle is not attended by any *positive* symptoms or physical signs except perhaps the presence of an exaggerated auricular wave in the cardiographic tracing. (See fig. 253).

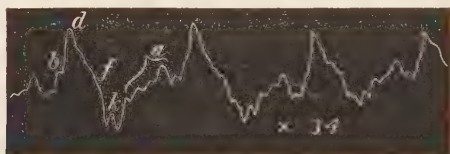


FIG. 253.—*Cardiographic tracing in a case of mitral stenosis.*—(After Galabin.)

‘Henry A., æt 8. Systolic and long, harsh presystolic murmurs at the apex, both accompanied by thrill. The presystolic murmur commenced immediately from the second sound, and was separated by a short pause from the systole. The bracket in the figure represents the duration of the murmur, which is separated by a distinct interval from the ventricular systole. The letter *a*, indicates the greatest auricular contraction.’¹—(*Guy’s Hospital Reports*, 1875, p. 314.)

Hypertrophy of the left auricle may be suspected, when the presence of mitral constriction is evidenced by a loud presystolic murmur, and when the lung symptoms, and signs of secondary changes in the right heart are slight. (The fact that the lung symptoms and signs of engorgement of the right heart are slight, does not necessarily show that the left auricle is hypertrophied, for a slight amount of mitral stenosis will of course only produce slight engorgement in the pulmonary circuit. A small amount of constriction is not, as a rule, attended by a well-marked presystolic murmur, for, as we have previously seen, a certain degree of constriction is required for the production of the fluid vein on which the murmur depends. If, therefore, a well-marked presystolic murmur is present, and

if, in addition, the symptoms are trivial, and if the signs of secondary changes in the right heart are slight, the probability is that the lesion is not altogether a trivial one. Hence it is allowable to conclude, in cases of this description, that the absence of lung symptoms, and of signs of right-sided hypertrophy, are due to compensating hypertrophy of the left auricle.)

Hypertrophy of the Right Auricle is much more rare than hypertrophy of the left, for tricuspid stenosis is, compared with mitral stenosis, a rare condition. The resisting power, too, of the right auricle, is less than that of the left, and the right auricle is, therefore, even more prone to undergo dilatation than the left. In some cases, however, a considerable degree of hypertrophy is observed. The best example which has come under my own notice is the case of primary lung obstruction (chronic bronchitis and emphysema from early childhood) to which I have previously referred. In that case not only was the right ventricle enormously hypertrophied and very little dilated, but the right auricle was also notably hypertrophied, and the hypertrophy of the right auricle seemed in that case to be due to constriction of the tricuspid orifice, which in its turn was apparently the result of a thickening of the muscular wall of the heart, and twisting of the tricuspid segments, which were themselves healthy. When the right auricle is hypertrophied as well as dilated, its pulsations can usually be perceived in the second and third right interspaces, but to this point I will again refer under the head of dilatation.

The Diagnosis of Hypertrophy of the Heart.

The diagnosis of hypertrophy of the ventricles, more especially of hypertrophy of the left ventricle, is, as a rule, easy, and is founded upon the fact that the physical signs of an enlarged and forcibly contracting heart are present. When the lungs are emphysematous, the diagnosis may be difficult or impossible, since it may be impossible to discover either

the enlargement or the increased force of the cardiac contractions. It must also be remembered that enlargement of the heart, on the one hand, may be simulated by all those conditions in which the præcordial dulness is increased; and that the increased action of a hypertrophied heart, on the other, may be simulated by the forcible contraction of temporary (neurotic) excitement. Before, therefore, diagnosing hypertrophy of the ventricles (either right or left or both), the observer must satisfy himself that both conditions, viz. an enlarged and a forcibly contracting heart are present; increased dulness without increased force of contraction, on the one hand, and increased action without increased dulness on the other, being insufficient. The opinion is, of course, greatly strengthened if a distinct (extra-cardiac or intra-cardiac) cause for the hypertrophy is present.

The reader is advised to refer to what has been stated with regard to the differential diagnosis of increased dulness over the præcordia, on page 129.

The differential diagnosis of pericardial effusion and hypertrophy of the heart is given in Table VII., page 334; while the differential diagnosis of hypertrophy and dilatation of the heart is discussed on page 626.

The conditions which are most likely to be confounded with hypertrophy of the ventricles are:—

1. *Increased exposure of the organ, or 'apparent enlargement,'* as it may be termed.—The points to which attention is to be directed in order to solve this question, are detailed on pages 131 and 132, to which the reader is again referred.

2. *Temporary over-action the result of neurotic causes (neurotic palpitation).*—In some cases the heart is, I believe, actually enlarged; but, in the majority, this is not so. Cardiac hypertrophy is to be excluded when the præcordial dulness is not increased, and when the apex beat is in its usual position. The exact characters of the pulse and of the cardiac contractions (whether slow and deliberate, quick and excited, etc.), must also be observed. In doubtful cases the presence of any intra-cardiac or extra-cardiac condition,

capable of producing hypertrophy, is a fact of the utmost importance.

3. *Displacement of the heart downwards and to the left.*—This condition may simulate hypertrophy of the left ventricle. The diagnosis is to be made by observing:—

(a) The force of the cardiac contractions—increased in hypertrophy, but not increased in simple displacement.

(b) The presence of any cause of displacement (such as a tumour or aneurism at the base of the heart) on the one hand, or of hypertrophy on the other (such as cirrhotic Bright's disease, valvular lesions, etc.).

(c) The outline of the percussion dulness—increased in hypertrophy, simply altered in position, when the heart is displaced.

The differential diagnosis of hypertrophy of the left and right ventricles is not always easy or possible, for the two conditions are frequently combined. Slight hypertrophy of the left ventricle may be masked by considerable hypertrophy of the right, and *vice versâ*. In well marked cases the distinction is easily made by attention to the following points:—

1. *The position of the apex beat and cardiac impulse.*—In hypertrophy of the left ventricle, the left apex beat is carried downwards and outwards, and the area of impulse is over the left heart; in hypertrophy of the right ventricle, the left apex beat may be displaced somewhat outwards, but not usually downwards, while the area of maximum impulse is over the lower end of the sternum and in the pit of the epigastrium, *i.e.* over the region of the right ventricle.

2. *The character of the impulse.*—In typical cases of hypertrophy of the left ventricle, the apex beat is well defined; the cardiac impulse is deliberate, forcible, and often has a heaving, pushing character. In hypertrophy of the right ventricle, the apex beat is, as a rule, more diffused than in health; the impulse is not deliberate, is less forcible than in hypertrophy of the left ventricle, and does not raise the chest wall *en masse*.

3. *The outline of the dulness.*—In hypertrophy of the left ventricle, the increased cardiac dulness extends both in the

vertical and transverse directions, but chiefly downwards and to the left. In hypertrophy of the right ventricle, the increase in the cardiac dulness is chiefly transverse, and it extends to the right as well as the left.

4. *The character of the radial pulse.*—In hypertrophy of the left ventricle, more particularly in those cases in which the hypertrophy depends upon extra-cardiac conditions, the radial pulse is firm and strong, and has the deliberate, forcible character possessed by the cardiac impulse. In hypertrophy of the right ventricle the radial pulse is usually small and weak. When the hypertrophy of the left ventricle depends upon aortic or mitral valvular disease, the firm, hard, sustained pulse characteristic of hypertrophy due to extra-cardiac conditions (such as cirrhotic Bright's disease) is not observed. The absence, therefore, of a hard sustained pulse does not exclude left-sided hypertrophy, but the presence of a small weak pulse, together with powerful pulsation over the right heart, is very suggestive of right-sided hypertrophy.

5. *The comparative loudness of the aortic and pulmonary second sounds.*—The aortic second sound is accentuated in those cases of hypertrophy of the left ventricle which depend upon extra-cardiac causes (when the hypertrophy is due to mitral or aortic valvular diseases, the accentuation is not, as a rule, present). The pulmonary second sound, on the other hand, is accentuated when the right ventricle is hypertrophied. It must, however, be remembered that an accentuated pulmonary second sound, although highly suggestive of hypertrophy of the right side, does not exclude hypertrophy of the left, for both forms of hypertrophy are often present in the same case, and may in fact be due to the same primary cause, —viz. mitral regurgitation.

Prognosis.—Hypertrophy is a compensatory and beneficial condition, in fact, nature's effort to meet a difficulty. It is not, however, an unmixed good, since it tends to produce atheroma, aneurism, arterial rupture, etc.

The prognosis of cardiac hypertrophy depends, therefore, for the most part, upon the cause of the hypertrophy. The

form of the hypertrophy (whether pure or combined with dilatation), and more especially the condition of the hypertrophied cardiac wall (whether undergoing fibroid or fatty changes) are also important points. In some cases, the cause of the hypertrophy can be removed, and it then subsides. In pregnancy and acute Bright's disease, for example, the hypertrophied left ventricle becomes quite normal with the disappearance of its cause. The same result also follows in cases of so-called idiopathic hypertrophy, a condition which is, I believe, present in some cases of long continued neurotic disturbance of the organ.

In other cases—and these constitute the large majority—the cause of the hypertrophy is permanent. When the primary lesion is a stationary one (*i.e.* non-progressive), when the hypertrophy is pure (*i.e.* unassociated with dilatation), when the cardiac muscle is healthy, and when the conditions for general and local nutrition are satisfactory, the prognosis is, on the whole, favourable. *Vice versâ* when the lesion is progressive, when the hypertrophy is combined with dilatation, when, more especially, the cardiac walls are degenerated, and when the conditions for local and general nutrition are unsatisfactory, the prognosis is bad. All these points have, however, been considered in great detail under the head of chronic valvular lesions, and to that description the reader is again referred.

Treatment.—From what has just been stated under the head of prognosis, the reader will perceive that, so long as the cause of the hypertrophy remains, the hypertrophy itself is to be sustained and encouraged, and the nutrition of the heart sustained and preserved. It is only in exceptional cases, as, for example, in cases of idiopathic hypertrophy, resulting from neurotic disturbance, that the hypertrophy itself is to be directly treated.

The indications for treatment in cases of cardiac hypertrophy are therefore:—

1. *To remove its cause.*—In the majority of cases of cardiac hypertrophy, the cause is organic and permanent, and does

not permit of removal. In a few cases, notably, for instance, in certain cases in which high arterial tension depends upon effete matters circulating in the blood, the excessive blood pressure can be removed, and the hypertrophy, which will certainly follow if the high arterial tension continues, prevented by careful regulation of the diet and attention to the excretions, more especially by the administration of mercurial and other purgatives and diuretics. Hypertrophy due to excessive muscular effort and to long continued neurotic excitement of the heart is, in many cases, curable.

2. *To reduce the strain on the heart as much as possible.*—In those cases in which the hypertrophy depends upon organic disease of a permanent and incurable character, it is important to reduce the strain on the heart, and to keep the circulation in a tranquil condition. The main objects of treatment are—to avoid everything which is likely to hasten the progress of the lesion on which the hypertrophy depends; and to maintain the general state of nutrition and the vitality of the cardiac muscle, so as to enable it to meet the extra demand which is required of it, and to compensate the lesion. In short, the whole object of treatment is to keep the primary lesion in a stationary or static condition, and to maintain just so much hypertrophy as is sufficient to balance the defect. In cases of this description it is particularly important to avoid all sudden causes of cardiac strain and excitement; in hypertrophy of the right ventricle, for example, whether the hypertrophy results from a mitral lesion or from a primary lung disease, acute pulmonary complications, such as attacks of bronchitis, which necessarily throw an increased and sudden strain on the right heart, must be carefully guarded against. Many other instances of a similar kind might of course be mentioned, and many other details of treatment given, but the subject has been so fully considered under the head of cardiac valvular lesions, that I must content myself with referring the reader to the previous portions of this work.

When any indications of cardiac failure arise, the administration of digitalis and other cardiac tonics and stimulants is

called for. In fact, the treatment which is appropriate for the early stages of dilatation of the heart, must then be carried out.

The occurrence of palpitation in cases of hypertrophy is usually, as I have previously insisted upon, an indication of commencing failure and of cardiac embarrassment. It is to be treated by reducing the strain on the heart, and by the administration of cardiac tonics, more especially digitalis. When the palpitation is due to neurotic causes, *i.e.* when it is not indicative of cardiac failure, cardiac sedatives are indicated. The application of cold, in the form of ice-bags, to the region of the heart; and the internal administration of aconite (drop doses every hour, carefully watched), of veratrum viride (five drops of the tincture three times daily), of digitalis and of hydrocyanic acid (two minim doses every three or four hours), are the most useful remedies. The application of a belladonna plaster to the præcordia also seems in some cases to be beneficial.

DILATATION OF THE HEART.

Definition.—Increased size of the cardiac cavities.

Varieties.—Three varieties of dilatation are usually described, *viz.* :—

1. *Simple dilatation.*—In this form the cardiac cavities or cavity—for one cavity only may be affected—are enlarged, and the muscular wall of the cavity retains its normal thickness. The total amount of muscular tissue surrounding the dilated cavity is necessarily increased, for otherwise it would be impossible for the wall of the cavity, the area of which is increased, to retain its normal thickness. Simple dilatation of the heart is therefore impossible, provided that the wall of the dilated cavity remains healthy; but since, in many cases, the muscular tissue is replaced by fibroid tissue, simple dilatation is possible, if we regard the amount of *muscular tissue in the cardiac wall*, and not the mere thickness of the wall itself. The distinction may perhaps seem a fine one, but it is not without practical advantages, for the muscular tissue of the

wall is the important element ; and it is essential in looking at the wall of a dilated heart, just as it is in looking at the wall of an apparently hypertrophied heart, to endeavour to determine whether it is composed of healthy muscular fibre or not.

2. *Dilatation with Hypertrophy*.¹—This condition, which used to be called *active* dilatation, is by far the most common form, so far, at all events, as the ventricles are concerned. The relative proportions of dilatation and hypertrophy vary indefinitely in different cases. When the hypertrophy is in excess or considerable, the condition is synonymous with eccentric hypertrophy ; when dilatation exceeds hypertrophy, the term *dilatation*, without any qualification, or *dilatation with hypertrophy*, is generally applied.

3. *Dilatation, with thinning or passive dilatation*, as it is sometimes termed.—This form is not so common as the preceding ones, but it is occasionally seen in the auricles, more especially the right auricle. As the term indicates, the cavity is dilated, and the wall thinner than natural. Cases of dilatation with thinning of the muscular tissue of the wall (if we regard the muscular fibres alone, irrespective of the endocardium, pericardium, and fibroid tissue and fat), are probably more common than is generally supposed.

Ætiology and Pathology.—Under normal circumstances the walls of the different cardiac cavities possess sufficient resisting power to withstand the pressure of the blood which they contain. When, however, the cardiac wall is weakened by disease, or when the blood pressure within the cavity is increased to such an extent that the existing power of the wall of the cavity is overcome, yielding or dilatation of the cavity will result. The great causes of cardiac dilatation are therefore in some cases vital, in others mechanical, viz. :—

¹ Under the head of dilatation with hypertrophy, those cases of so-called simple dilatation (*i.e.* dilatation with normal thickness of wall) in which the wall of the dilated cavity is composed of muscular tissue, ought, strictly speaking, to be included ; for, in cases of this description, the total amount of muscular tissue in the wall of the affected cavity is increased.

Ætiology and Pathology of Cardiac Dilatation. 611

1. *Diminished resisting power of the cardiac walls.*—All conditions, which impair the vitality and integrity of the muscular tissue, impair, of course, the resisting power of the cardiac wall. In some cases, the structural change in the cardiac muscle is quickly established, *i.e.* is acute ; under this head are included acute fatty degeneration, acute myocarditis, acute ulcerative endocarditis, and the muscular changes (cloudy swelling and softening) which are met with in the continued fevers. In other cases, the muscular lesion is slowly established ; in this group are included fatty infiltration, fatty degeneration, fibroid degeneration, the relaxed and debilitated condition of the cardiac muscle, which is associated with anæmia, impaired general nutrition, conditions of nervous depression and general muscular relaxation. The conditions, therefore, which produce the structural changes in the cardiac wall leading to diminished resistance, and therefore to dilatation, are in some cases local and in others general ; the fibroid degeneration which results from myocarditis, is an example of the former ; the fatty degeneration, associated with general anæmia (chlorosis and progressive pernicious anæmia) of the latter.

When the resisting power of the cardiac wall is much impaired, dilatation may result under the normal blood pressure. When the lesion of the muscle is a slight one, an abnormal degree of blood pressure may be required to produce dilatation ; and, as a matter of fact, both causes are frequently combined. The lesion, which produces the diminished resistance, may be limited to a particular part of the wall of one cardiac cavity—the left ventricle, for example—the dilatation which results will, of course, be local, and the condition is then synonymous with that which has been previously described under the term *aneurism* (partial or local aneurism) *of the heart*. In other cases, the whole wall of one cavity is affected, and the whole of that cavity becomes dilated ; to these cases the term *partial dilatation of the heart* is sometimes applied.¹ In others

¹ Partial dilatation *of the heart* must not be confounded with partial or local dilatation of *one cavity*, *i.e.* with the condition which is more appropriately termed aneurism of the heart.

again the dilatation affects all the cavities of the heart ; it is then said to be *general*.

2. *Increased blood pressure within the heart.*—Increased blood pressure is, of course, a mechanical cause of dilatation ; and other things being equal, the greater the pressure the greater the dilatation. It must not, however, be supposed that the problem is entirely a mechanical one. Several important considerations, more especially the rapidity with which the increased pressure is established, the vitality of the cardiac muscle, the reserve force possessed by the wall of the particular cavity on which the increased pressure is exercised, and the condition of the cardiac wall (whether relaxed or contracted) at the time when the pressure is applied, all exert an important influence on the result.

(a) *The rapidity with which the pressure is established and the vital condition of the cardiac muscle.*—When the increased pressure is quickly established, and when that pressure is in excess of the resisting power (*i.e.* of the reserve force) possessed by the cardiac wall on which it is brought to bear, dilatation must necessarily result.—I do not, of course, refer to those cases in which the excessive pressure is so great as to produce paralysis of the cardiac wall, in such cases death would of course result ; nor to those cases in which the pressure is of merely temporary duration, temporary over-distention of the right heart is of frequent occurrence, and is probably relieved by the yielding (safety-valve action) of the tricuspid valve, which may, of course, occur even when the muscular tissue of the cardiac wall is perfectly healthy. One of the best examples of dilatation produced in this manner is that which follows the sudden rupture of an aortic valve segment.

But even in cases of this description the dilatation is not pure (*i.e.* unattended with hypertrophy), for although the dilatation always remains in excess—and usually largely in excess—a certain amount of hypertrophy is gradually established. *Provided that the cardiac muscle is sound*, it is almost impossible to have pure dilatation. Should the patient survive a sufficient length of time, some increase of

the muscular wall, *i.e.* some hypertrophy, will almost certainly occur; the dilated cavity contains an excessive quantity of blood, its muscular wall is consequently over-stimulated, and, provided that its muscular fibre is healthy, the increased strain which is thrown upon it, and the excessive effort which is put forth in its attempts to expel the abnormally large quantity of blood which it contains, will almost of necessity result in the production of some hypertrophy. When, therefore, the blood pressure within the heart is quickly raised beyond the resisting power of the cardiac wall, dilatation is necessarily produced, but the walls of the dilated cavity tend at the same time to become hypertrophied. As a matter of fact, however, the dilatation almost invariably remains in excess, and, as a rule, largely, in excess of the hypertrophy.

When, on the other hand, the increased pressure is very slowly and gradually established, hypertrophy results, provided, of course, as has been previously explained, that the vitality of the cardiac muscle is good and it is able to respond to the increased strain which is thrown upon it. In cases of this description, the pressure is in fact never in excess of the reserve force, for, as the pressure slowly and gradually increases, the resisting power keeps pace with it, in consequence of the hypertrophy, which is *pari passu* established. The hypertrophy of the left ventricle, which accompanies the cirrhotic form of Bright's disease, is a typical example in point.

When, however, the cardiac walls are degenerated, increased pressure produces dilatation, however slowly and gradually it is established. And even in those cases in which the muscular fibre was originally sound and the hypertrophy for a time pure, dilatation often ultimately follows in consequence of degenerative changes, which are so apt to occur in the walls of a hypertrophied heart.

We may say, then, that a dilated heart, whose muscular walls are healthy, tends to become hypertrophied, while a hypertrophied heart tends ultimately to become dilated.

(b) *The cavity on which the increased pressure is exerted.*—The weaker the wall the greater the tendency to dilatation; hence it is that the auricles, the walls of which possess little

reserve force, are more liable, other things being equal, to become dilated than the ventricles, and the cavities of the right heart than those of the left. The order then in which the cardiac cavities tend to become dilated is as follows :—

Right auricle.

Left auricle.

Right ventricle.

Left ventricle.

The clinical and pathological conditions, which produce increased pressure within the cardiac cavities, and which may therefore lead to dilatation, are very various. The more important have been mentioned in speaking of the causes of hypertrophy. Increased tension in the left ventricle, during its systole, is produced by all those conditions which interfere with the passage of the blood through the arterial and capillary systems, or which cause obstruction at the aortic orifice. (These conditions are, as we have previously seen, more likely to produce hypertrophy than dilatation.) Aortic incompetence and mitral incompetence produce over-distention of the left ventricle during its diastole, and are fertile sources of dilatation of that cavity.

Mitral stenosis and mitral incompetence both produce increased tension in the left auricle (the former during systole, the latter during diastole), and both cause dilatation of that cavity. Aortic lesions and increased tension in the arterial system may ultimately lead to the same result.

Over-distention of the right ventricle, during its systole, is produced by all conditions which interfere with the blood-flow through the lungs (primary lung disease, mitral lesions, etc.), or which cause an obstruction at the pulmonary orifice or in the pulmonary artery. Tricuspid incompetence produces over-distention of the right ventricle during its diastole. All of these conditions cause dilatation. (Pulmonary incompetence is so extremely rare, that for practical purposes it may be left out of account.) Increased tension of the blood in the right auricle is usually due to some obstruction in front of the right ventricle, or to tricuspid incompetence, or to both conditions combined. Tricuspid stenosis of course produces the

same result, *i.e.* over-distention and dilatation of the right auricle.

(c) *The condition of the cardiac wall (whether flaccid or contracted) at the time when the pressure is exercised.*—Forcible dilatation of the cardiac wall when it is in a flaccid and enlarged condition, *i.e.* during its diastole, is much more likely to produce dilatation than forcible pressure when the wall is contracted, *i.e.* during the systole. (This statement does not, however, apply to those cases in which a limited portion of the cardiac wall has lost its resisting power, in consequence of degenerative changes.) It is probably in consequence of this fact that the cavity of the left ventricle becomes dilated in aortic and in mitral incompetence. In the former case, *i.e.* aortic regurgitation, blood is poured into the cavity from two sources, *viz.* from the left auricle through the mitral in the normal, and from the aorta in the reverse course of the circulation; the left ventricle becomes more quickly and more forcibly distended than in health, and before the occurrence of each systole it is, as it were, dilated. In the latter case, *i.e.* mitral incompetence, the blood stream, which passes through the mitral orifice is a larger and more forcible stream than in health, in consequence of the excessive blood pressure in the parts behind the affected (*i.e.* mitral) orifice.

During their systole, on the other hand, the walls of the heart are able to withstand high internal pressure without the production of dilatation. The pressure within the left ventricle, for example, during the systole of a normal heart, is extremely great; it is still greater in cases of aortic stenosis in which the blood-flow from the cavity is obstructed and the wall of the cavity hypertrophied; and yet the hypertrophy is in many cases pure, *i.e.* unattended with any dilatation. When, therefore, the increased pressure is brought to bear during systole, the tendency to dilatation is comparatively slight. But since these points have been fully explained, when treating of the different valvular lesions, I must refer the reader to what has been already said on those subjects for further details. In cases of this nature local dilatations or partial aneurisms of the heart are, as we have previously

seen, produced, the weakened part of the cardiac wall yielding to the very great internal pressure produced during the cardiac systole.

3. *Traction from without.*—Theoretically this is a possible cause of cardiac dilatation; and, as a matter of fact, we find that in many cases of adherent pericardium cardiac dilatation is actually present. In some of these cases the dilatation is undoubtedly due to other conditions (more especially associated valvular lesions) and not to the adherent pericardium; in other cases in which the cardiac valves are healthy, but the pericardium adherent, the dilatation is chiefly due to structural changes in the cardiac muscle which are so frequently present; traction from without is probably, in part at least, the cause of the dilatation in some cases of adherent pericardium. When, on the contrary, the muscular walls are healthy, pericardial adhesions, by the resistance which they offer to the free contraction of the organ, are apt to produce hypertrophy. In short, the result (whether hypertrophy or dilatation) which is produced by pericardial adhesions depends, in my opinion, chiefly upon the vitality, so to speak, of the cardiac wall, and upon the part of the heart upon which the traction is exercised. When the cardiac wall is healthy, hypertrophy will probably result. When, on the contrary, the cardiac wall is degenerated, dilatation will probably be produced (other things being equal). Traction on the wall of the right ventricle is more likely to produce dilatation than traction on the wall of the left, for the reserve force, and therefore the tendency to hypertrophy, is greater in the left ventricle than in the right.

Both results (hypertrophy and dilatation) are more likely to occur when the pericardial sac is adherent to the parts external to it (sternum, lungs, etc.), as well as to the surface of the heart. The more extensive the adhesions the greater the tendency both to hypertrophy and to dilatation.

Dilatation is said to occur sometimes in health as the result of violent muscular efforts or other sudden causes of cardiac over-distention; but whether such a result does ever occur in *perfect* health is, in my opinion, extremely doubtful.

Doubtless over-distention, of the right heart more particularly, is of frequent occurrence, and it may be granted that if such over-distention were frequently repeated, some permanent dilatation would probably remain. Occasional over-distention could hardly, I think, lead to permanent dilatation unless the resisting power of the cardiac muscle were lowered, in consequence of some general constitutional cause or local structural defect.

The naked eye appearances which a dilated heart presents (its size and weight, shape, etc.) vary of course with:—the part of the organ which happens to be affected, the extent of the dilatation, the condition of the pericardium, the amount of sub-pericardial fat, and the state of the cardiac muscle, whether flaccid, as the result of fatty or other changes, or indurated as the result of combined hypertrophy and fibroid degeneration. It is unnecessary to go into details. The microscopical appearances, too, are very varied. In some cases the muscle is fairly healthy; in others, fatty and fibroid changes are present; in others, again, cloudy swelling, or acute myocarditis is observed. All of these conditions are described in detail in other parts of the work, and need not be repeated.

Pathological Physiology.—Dilatation, which is the direct opposite of hypertrophy, inasmuch as it impairs the efficiency of the cardiac pump, is an injurious condition. But while this statement may be safely laid down as a general proposition, there are some circumstances in which a certain degree of dilatation is beneficial and compensatory. In regurgitant valvular lesions (say mitral regurgitation, for example) dilatation of the cavity which is situated behind the affected orifice (*i.e.* the left auricle in the case we are considering) is beneficial, provided that it is just sufficient to accommodate the blood which regurgitates at each systole, and provided that the wall of the dilated cavity (*i.e.* of the left auricle) is at the same time sufficiently hypertrophied to completely empty the cavity, *i.e.* to expel into the cavity of the left ventricle all the blood which has regurgitated, together with the normal quantity of blood which reaches it from the pulmonary veins. Such a

perfect balance of compensation is, however, rarely seen; and, speaking generally, it may be safely affirmed that dilatation is a baneful condition; blood tends to stagnate in the dilated cavity, the circulation in the parts behind becomes retarded, the supply of blood to the parts beyond, defective; in short, a condition of arterial anæmia and venous engorgement, with all the secondary results and complications, which I have fully described in speaking of mitral and tricuspid valvular lesions, tends to become established. When the dilated cavity is a ventricle, relative incompetence of the auriculo-ventricular valve connected with it is also apt to be produced. Dilatation of the left ventricle is apt to produce incompetence of the mitral, and dilatation of the right ventricle of the tricuspid orifice. Clots are apt to form in those parts of the cardiac cavities in which the blood current is sluggish or stagnant (the appendices of the auricles, apices of the ventricles, etc.) Portions of these clots may become detached and carried as embolic infarctions to distant parts. In rarer cases, alarming symptoms and death result from the presence of thrombi within the heart itself. To sum up, in dilatation a greater amount of blood requires expulsion from the dilated cavity; the walls of the cavity have less power than normal; in addition, valvular incompetence (mitral and tricuspid regurgitation) is often established; clots may form in the dilated cavity, and embolic plugging of distant vessels may be produced.

Symptoms.—The symptoms which characterise dilatation of the heart are essentially those of cardiac weakness, engorgement of the venous, and anæmia of the arterial, circulations. Differences in detail are, of course, observed in accordance more particularly with the seat of the lesion (*i.e.* whether the left or the right heart is affected), the extent of the dilatation, and the associated pathological conditions (degree of hypertrophy which accompanies the dilatation, the presence of associated valvular lesions).

In advanced cases of dilatation of the left heart, the symptoms are identical with those of advanced mitral lesions. (See

page 439.) In advanced dilatation of the right heart, the symptoms correspond to those of tricuspid regurgitation. (See page 534.)

Physical signs.—The physical signs which are met with in dilatation of the heart vary, of course, with the nature of the primary lesion on which the dilatation depends, and the particular part of the organ which is dilated. The physical signs characteristic of dilatation *per se* (*i.e.* irrespective of the primary lesion on which the dilatation depends), are indicative of enlargement and weakness of the heart. Here, as in the case of hypertrophy, it is essential to remember that the physical signs indicative of the size of the heart and the strength of the cardiac impulse, are materially modified by the condition of the lungs. When the lungs are emphysematous, it may be difficult or impossible to detect, by means of percussion, enlargement of the organ. (This statement applies more especially to enlargement of the left side.) When, again, the lungs are very voluminous, the force of the cardiac impulse as felt by the hand over the præcordia, is diminished, and the conduction of the cardiac sounds to the ear interfered with; under such circumstances the heart may be acting with quite the normal strength, but may appear to be weak. An emphysematous condition of the lungs adds, therefore, considerably to the difficulties of diagnosing a dilated heart; for, on the one hand, it may be difficult or impossible, when the heart is actually dilated, to detect the enlargement by means of percussion, and, on the other, a perfectly normal or even hypertrophied heart may appear (owing to the feeble impulse and feeble sounds) to be weak.

In conditions of general dilatation, the impulse both of the left and right hearts is weaker than in health; the apex beat is feeble, or altogether imperceptible. When the right ventricle is dilated and the left normal, the impulse over the right heart is feeble or imperceptible; the impulse of the left heart may be of normal strength. It must, however, be remembered that when the right ventricle is dilated, it may be difficult or impossible to distinguish the impulse of the left heart, for the

heart is rotated on itself, the right ventricle making its way in front of the left. The impulse, when it can be felt, is usually more diffused than in health, and consists of a short slap or tap, indicative of a quick, short, irritable and feeble ventricular contraction, the exact opposite of the prolonged, steady, deliberate heave of hypertrophy. The cardiac action is often irregular; and the frequency of the cardiac contractions is very generally increased.

The position of the apex beat varies in accordance with the part of the heart which is dilated. In dilatation of the left ventricle, the left apex beat can usually be felt (for the dilatation is generally combined with some, often with considerable, hypertrophy), and is displaced downwards and to the left; when the right ventricle is dilated, the left apex beat is often effaced, owing to the fact that the right ventricle makes its way in front, displacing the left heart backwards and to the left; when the right heart is much dilated, pulsation can often be seen and felt in the second left interspace. In some cases this pulsation is, I think, derived from the conus arteriosus of the right heart; in others, from a dilated pulmonary artery. When the left auricle is very much dilated, as it is in some cases of mitral stenosis, for example, its pulsation can sometimes, it is said, be seen and felt in the second left interspace. I am not prepared to say unconditionally that this auricular pulsation does not occur, but I have never seen any *post-mortem* evidence in support of it. When the right auricle is much dilated, pulsation sometimes can be felt in the third right interspace adjacent to the sternum. When the tricuspid is incompetent, as it very generally is in cases of this description, the pulsation is systolic in rhythm, and accompanied by true venous pulsation in the neck and often also in the liver.

The area of percussion dulness (both superficial and deep) is increased (except as previously mentioned in conditions of emphysema); the direction of the increase varies in accordance with the particular part of the heart which is dilated. (See figs. 246 and 252.) When the left ventricle is affected, the increased dulness extends both in the vertical and transverse directions, and is chiefly downwards and to the left; when

the right ventricle is dilated, the increased dulness is for the most part transverse.

When the ventricles are dilated, the duration of the *first* sound is shorter than in health, its pitch higher ; it is in fact more valvular and less muscular than the normal first sound ; in many cases it is with difficulty distinguished from the second sound. In advanced cases of dilatation, more especially dilatation of the right ventricle, the first sound is extremely weak or almost imperceptible, and is often replaced by a systolic murmur due to mitral or tricuspid regurgitation. When the dilatation is limited to the right ventricle, the sounds of the left heart may be normal, and *vice versâ* should the dilatation be limited to the left ventricle (a condition which is rather of theoretical than practical importance, for dilatation of the left ventricle is almost always associated with some dilatation of the right), the first sound derived from the right heart might be normal.

The character of the *second* sound varies considerably in different cases. In dilatation of the left ventricle, the result of mitral regurgitation, for example, the aortic second sound is weaker, the pulmonary second sound louder than in health ; in dilatation of the left ventricle, the result of aortic regurgitation, the aortic second sound is replaced by a murmur, the pulmonary second sound is either normal or accentuated. When the right ventricle is dilated, the pulmonary second sound is generally accentuated, and the aortic second sound usually weaker than normal. (Accentuation of the pulmonary second sound is very generally present in the earlier stages of right-sided dilatation due to obstruction in front ; in the final stages, when the dilatation is considerable, the tricuspid incompetent, and the right ventricle injecting little blood into the pulmonary artery, the pulmonary second sound is much less loud than in the earlier stages of the case.) These variations, which depend, of course, upon the condition of the aortic and pulmonary valve segments, the amount of blood which is being injected into the aorta and pulmonary artery respectively, and the resistance in the arterial and pulmonary circulations, will be readily understood, if the reader will again

refer to the details given in the earlier parts of this work. (See pages 145 and 146.)

The systolic portion of the *cardiograph tracing* is unsustained, the summit pointed, and the up-stroke, which represents the commencement of the ventricular systole, followed by a rapid fall. (See fig. 254.)

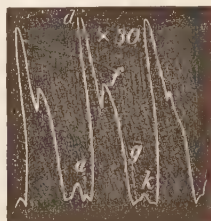


FIG. 254.—*Cardiogram in a case of exophthalmic goitre.*—(After Galabin.)

‘Rebecca S., æt. 20. Mitral regurgitation combined with exophthalmic goitre. Heart dilated and hypertrophied. Pulse 110.’—(*Guy’s Hospital Reports*, 1875, p. 314).

The radial pulse in cardiac dilatation is, as a rule, quicker than in health, small, weak, and very often irregular or intermittent. In extreme conditions of dilatation of the left ventricle, the frequency of the pulse is often less than the frequency of the cardiac contractions, many of the pulse waves being too feeble to reach the wrist. When the dilatation is combined with much hypertrophy, as it usually is in aortic regurgitation for example, the characters of the radial pulse are of course quite different.

Diagnosis.—The diagnosis of dilatation of the heart is more difficult than the diagnosis of cardiac hypertrophy, for there are several conditions which may simulate an enlarged and *weak* heart—notably effusion into the sac of the pericardium—but few which are likely to be confounded with an enlarged and *strong* (i.e. a hypertrophied) heart.

Given the presence of increased dulness over the præcordial region, the steps in the diagnosis of cardiac hypertrophy and cardiac dilatation are the same up to a certain point:—

Step No. 1.—*Is the increased dulness over the præcordium*

derived from the heart itself, or is it due to the presence of some non-resonant substance in contact with the organ?

As a rule there is little difficulty in coming to a correct conclusion on this point. As we have previously seen, the chief pathological conditions which give rise to dulness in the neighbourhood of the heart, are:—

(a) Consolidations of the adjacent portions of the lungs (apoplectic, pneumonic, tubercular, sarcomatous, etc.)

(b) Fluid in the pleural cavity.

(c) Enlargement of the liver.

(d) Tumours, collections of fat, or inflammatory deposits in the anterior mediastinum.

(e) Aneurism of the first portion of the aortic arch.

Now in most of these conditions—in the vast majority of cases met with in practice—the increased dulness is not confined to the limits of the præcordia, but extends often for a considerable distance into the surrounding regions of the chest.¹ In many cases too, it does not conform to the shape of the dulness which results from an enlargement of the heart or pericardium.²

There are, too, as a rule, other symptoms and physical signs indicative of the cause of the dulness. In consolidations of the lung, for example, cough, expectoration, and alterations of the respiratory murmur over the seat of the dulness (tubular breathing, râles, etc.), would probably be present.

In addition to those positive facts, the negative evidence—that there are no signs nor symptoms of disease of the heart or pericardium—confirms the diagnosis.

The points, then, to which attention should be directed, in

¹ Dulness, resulting from a limited consolidation of those portions of the lungs in contact with the heart, might of course be limited to the præcordia. In such a case the other physical signs and symptoms (negative and positive) would be quite sufficient to determine the diagnosis.

² It would be extremely difficult, indeed in many cases impossible, to distinguish the dulness due to a small tumour or inflammatory accumulation in the anterior mediastinum, from the dulness which results from enlargement of the heart or pericardium. Fortunately limited tumours of this description are rare, and the difficulty in diagnosis is therefore seldom met with in practice.

order to come to a conclusion as to the first step in the diagnosis, are :—

1. The extent and outline of the dulness.
2. The presence of symptoms or physical signs indicative of disease of the adjacent parts.
3. The condition of the heart and pericardium, as determined by other methods of investigation.

Step No. 2.—If the dulness is directly derived from the heart itself, does it result from increased exposure (i.e. apparent enlargement), or from actual increase in the size of the organ?

By far the most common cause of ‘increased exposure’ of the heart is retraction of the anterior margins of the lungs, a condition which usually results from pleurisy or cirrhosis.

In seeking then to decide whether the increased dulness is due to apparent or actual enlargement of the organ, attention must be particularly directed to the condition of the lungs. A history of previous pleurisy; the fact that the anterior margins of the lungs are fixed by adhesions, and do not expand and cover up the heart during inspiration—a point which can be determined by percussion and auscultation, during inspiration and expiration respectively; or, the presence of symptoms and signs of cirrhosis, phthisis, etc., would of course be in favour of increased exposure (apparent enlargement)—an opinion which would be confirmed by the absence of symptoms and signs of pericardial or cardiac disease, or of any extra-cardiac cause of enlargement of the heart, such as cirrhosis of the kidney or atheroma.

But while these are the points to which attention should be directed, in order to make a diagnosis, it must be confessed that a positive opinion cannot always be arrived at; and it is still more difficult to exclude any enlargement of the heart itself, in those cases in which the increased exposure is due to forward displacement of the organ. In many of these cases the heart is actually enlarged as well as displaced; and in those cases in which there is no enlargement of the organ, the strong cardiac impulse which may be very noticeable, and the presence of intra-cardiac murmurs, which may be produced by pressure alterations at the valvular

orifices, may make it impossible to exclude all cardiac hypertrophy. In cases of this description therefore (which, as I have previously remarked, are extremely rare), a positive opinion that the increased dulness is due to forward displacement, and not to enlargement of the heart itself, could only be ventured upon when :—

(a) There is distinct evidence of an aneurism or tumour behind the heart, *i.e.* of the presence of an efficient cause of forward displacement.

(b) There are no signs nor symptoms of cardiac disease ; and no extra-cardiac cause of enlargement, such as chronic Bright's disease.

Step No. 3.—If the dulness is due to an actual increase in the size of the organ, does it result from fluid in the sac of the pericardium, or from enlargement of the heart itself?

The differential diagnosis of pericarditis with effusion, and of hypertrophy of the heart, seldom presents any difficulty. The chief points of distinction are given in Table VII., p. 334.

The differential diagnosis of dilatation of the heart and pericarditis with effusion, is often most difficult, but can usually be determined by attention to the outline of the cardiac dulness, the position of the cardiac impulse, and its relation to the left apex beat, the presence or absence of pericardial friction, the presence or absence of fever, the history of the case, and the associated diseased conditions. The points of distinction are considered in detail in Table VI., p. 333.

Step No. 4.—If the increased dulness is due to enlargement of the heart itself, is the enlargement general or partial, and is the heart hypertrophied or dilated?

In trying to determine whether the enlargement of the heart is general or partial, and what particular cavity or cavities are affected, we observe :—

1. *The cause of the enlargement.*—When, for example, the cause of the enlargement is incompetence of the aortic valves, we know that the left ventricle must be affected, and that the

left auricle and right side are, in many cases, affected in the later stages of the case. If, again, chronic bronchitis and emphysema are the primary cause of the cardiac enlargement, the right cavities are first and most affected, although, as I have previously pointed out, the left ventricle may subsequently become hypertrophied.

2. *The position and character of the cardiac impulse and apex beat.*—When, for example, the left ventricle is hypertrophied, the impulse and apex beat are well defined, and are displaced downwards and to the left. When the right ventricle is affected, the impulse is carried downwards and to the right. It is, however, unnecessary to go into details, since all of these points have been fully considered in describing hypertrophy and dilatation of the different cardiac cavities.

3. *The position and outline of the cardiac dulness.*—This, as will at once be understood from what has been stated in describing the physical signs of hypertrophy and dilatation, is an extremely important means of determining the part of the heart which is enlarged.

4. *The character of the radial pulse.*—(See physical signs of hypertrophy and dilatation.)

5. *The comparative degree of intensity of the aortic and pulmonary second sounds.*—(See physical signs of hypertrophy and dilatation.)

The differential diagnosis of hypertrophy and dilatation.—Hypertrophy and dilatation are, as I have so frequently stated, very generally combined ; in many cases, therefore, the heart cannot be said, in strict language, to be hypertrophied or dilated, for it is both. As a matter of practical convenience, however, we are in the habit of terming an enlarged heart, in which hypertrophy predominates considerably over dilatation, a hypertrophied heart, and *vice versâ* an enlarged heart in which dilatation is the most conspicuous condition, we term a dilated heart, even although it may at the same time be to some extent hypertrophied. The reader, who has attentively studied the physical signs detailed under the head of hypertrophy and dilatation respectively, will have no difficulty in

distinguishing a case of pure hypertrophy from a case of pure dilatation. I need not, therefore, repeat the points of distinction between the two conditions. I must, however, again insist upon the necessity of taking into account the condition of the lungs, and not mistaking an enlarged heart which is extensively overlapped by an emphysematous lung for an enlarged weak (*i.e.* dilated) organ.

Prognosis.—Speaking generally, it may be stated that dilatation is an unfavourable condition, and that the prognosis of dilatation is bad. Each case must, however, be judged on its own merits. The most important point in trying to form an opinion as to the future progress of the case, is to determine the exact cause of the dilatation. If the cause can be removed, the dilatation, in many cases, disappears, and a cure is effected. The dilatation, which is due to the fatty degeneration of chlorosis, is almost certainly curable; that met with in progressive pernicious anæmia can, in many cases, also be completely removed by treatment. Dilatation and embarrassment of the right heart are often seen as temporary results of bronchitis. It is, however, unnecessary to quote further examples. Let me, however, again insist upon the fundamental importance of trying to determine the exact nature of the cause of the cardiac lesion, and the exact structural condition of the cardiac muscle. It is only after having satisfied himself on these points that the observer is in a position to give an intelligent opinion as to the future progress of the case. The complications and associated pathological conditions are, of course, also to be carefully investigated, and all the other points which have been detailed in speaking of the prognosis of cardiac valvular lesions attended to. In short, the physician must here, as in every other disease, endeavour to take a broad, comprehensive, and all-round view of the case.

Treatment.—The treatment of cardiac dilatation is practically identical with the treatment of cardiac valvular lesions. The *first* indication is to endeavour to remove the cause—whether it be some structural change in the cardiac muscle, or

a mechanical cause of increased intra-cardiac tension, or both. In the *second* place, the organ must, so far as possible, be placed at rest, and all causes of increased vascular tension (both in the systemic and pulmonary circulations), and of cardiac excitement, carefully avoided. In the *third* place, cardiac tonics and stimulants must be administered in accordance with the needs of each individual case. In every case, the general health is to be maintained in the highest possible state of efficiency. In the *fourth* place, engorgement of the venous system, and all its secondary results, whether in the lungs or organs behind the right heart, must be treated in the manner recommended in speaking of mitral lesions. (The treatment of many cases of dilatation is identical with the treatment of mitral lesions. See page 463.)

ATROPHY OF THE HEART.

Definition.—An atrophied heart may be defined as a heart which is decreased in weight and often also in size, in consequence of wasting or atrophy of its muscular fibres.

This definition does not include all cases in which the muscular tissue of the heart is wasted; in many cases of fibroid degeneration and fatty infiltration, for example, the muscular fibres are in places wasted, but the weight of the heart is, as a rule, fully up to or even beyond the average; in cases of this description the atrophy is, as a rule, local and limited in distribution, though cases of fatty infiltration are occasionally met with in which the greater part of the muscular tissue throughout the heart is atrophied and replaced by fat.

There are, in short, two distinct forms of cardiac atrophy. In *one*, which includes the cases embraced in my definition, the atrophy is part and parcel of a general atrophy; in this form the whole organ is affected, though the wasting may be more advanced in the left than in the right heart. In these cases there is no disease of the heart itself.

In the *second* form, the atrophy is, as a rule, localised, and is the direct result of some pathological change in the

heart itself, such as fibroid degeneration, fatty infiltration, fatty degeneration, disease of the coronary arteries. In the former there are, as we shall presently see, no cardiac symptoms; in the latter, cardiac symptoms are usually prominent. Cases included in the latter group are not covered by the following description, for, as I have already remarked, it is seldom that in them the weight of the heart is below the average.

Ætiology and Pathology.—Atrophy of the heart is sometimes congenital, but much more frequently acquired.

The *congenital form* is more common in women than in men, and is usually associated with imperfect development of the aorta and other parts of the vascular apparatus; the general growth of the system is usually retarded, and the sexual organs often in a rudimentary condition.

The chief points which distinguish the congenital from the acquired form of atrophy are:—

(1) The fact that the diminished size of the heart is not necessarily associated with general emaciation.

(2) The condition of the pericardial covering and of the blood vessels on the exterior of the organ. In the congenital form the myocardium fills, as it were, its pericardial covering, and the vessels on the exterior of the heart differ in no way from those of the normal organ. In the acquired form, on the contrary, the muscular substance is too small, so to speak, for its vascular and pericardial coverings, for fibrous tissue and vessels do not atrophy to the same extent as muscular tissue; the pericardial covering, more especially at the apex of the organ, is thrown into wrinkles and folds, and the vessels on the surface of the atrophied organ stand prominently out, and are much more tortuous than normal. In the congenital form, again, the usual amount of sub-pericardial fat is present, whereas in the acquired form it is generally much diminished, or has entirely disappeared.¹

¹ It must, of course, be remembered that in many persons, more especially in young subjects, there is little or no fat on the exterior of the healthy, normal, heart.

The acquired form of atrophy, included in the definition given above (*i.e.* in which the weight of the heart is decreased), is usually the result of some general constitutional state or local disease which very slowly and gradually produces emaciation, diminishes the total amount of blood in the body, and precludes active bodily exercise. The conditions which, above all others, fulfil these requirements, are stricture of the œsophagus and cancer of the pylorus. Patients affected with these diseases often die slowly from inanition, after lying at rest in bed for long periods of time. Cancer of the pylorus is, in fact, as Louis long ago pointed out, the disease in which the heart becomes smaller than in any other condition.

In many cases of phthisis, too, the heart weighs less than normal, and is distinctly atrophied; the wasting is never, however, so great as it is in cancer of the pylorus, for in those cases which run a very chronic course, *i.e.* cases of fibroid phthisis, the obstruction to the passage of the blood through the lungs necessitates over-action on the part of the right ventricle, in consequence of which the weight of the heart is to a large extent maintained; and in cases which run a rapid course, the patient does not live sufficiently long to permit of extreme atrophy taking place; the same cause, too, which prevents atrophy in chronic cases, *viz.*, over-action of the heart, is also present in acute cases, though in a less degree. (In acute cases there is not so much obstruction to the passage of the blood through the lungs as in cases of fibroid phthisis, but another factor which necessitates over-action comes into play, *viz.* increased frequency of contraction, and it is more marked, the more rapid the progress of the case.)

In diabetes mellitus, in cases of prolonged suppuration, and in many other chronic conditions in which there is much emaciation, the heart shares in the general wasting. In uncomplicated cases of waxy disease of the kidneys, the heart is usually smaller than normal, a point of some interest, when it is remembered that in other varieties of Bright's disease, more especially in the cirrhotic form, the opposite condition, *viz.* hypertrophy, occurs.

The essential characteristic of an atrophied heart, using the

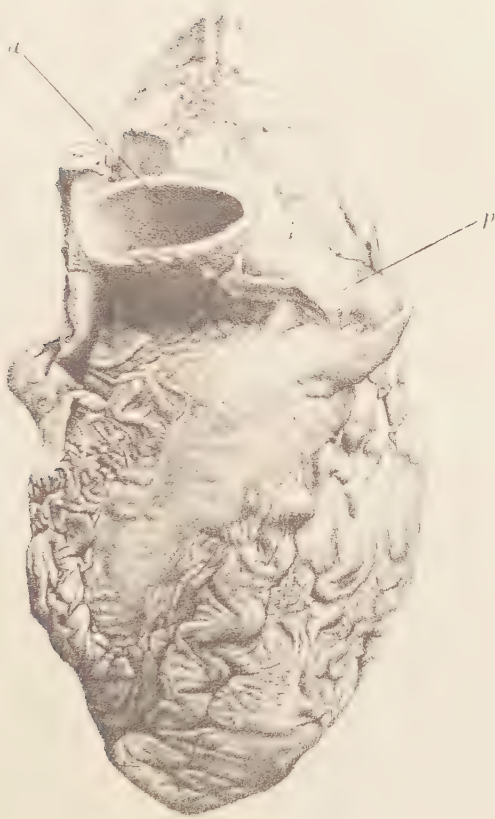


FIG. 255.—*Atrophy of the heart, seen from the front. (Exact size.)*

The heart is suspended by the auricle. The patient, a woman *æt.* 45, died after a very lingering illness from cancer of the pylorus. When in health she was well nourished though never fat; her height was 5 feet 4 inches.

Weight of the heart=2 oz., 12 drs., 11 grs. (Av.).

a, points to the aorta; *p*, to the pulmonary artery.

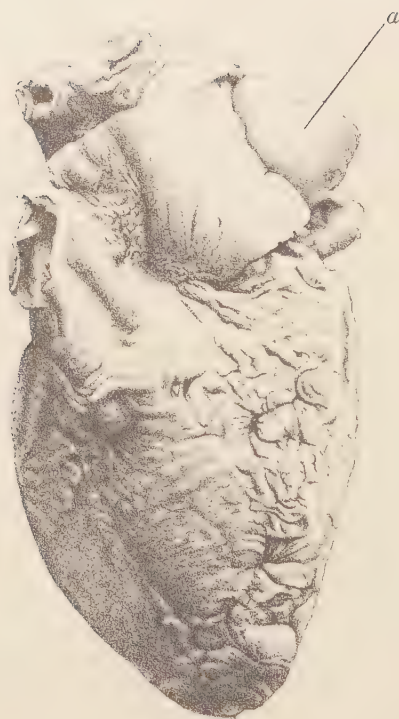


FIG. 256.—*Atrophy of the heart, seen from behind. (Exact size.)*

The patient, a woman *æt.* 45, died after a very lingering illness from cancer of the pylorus. When in health she was well nourished, though never fat; her height was 5 feet 4 inches.

Weight of the heart=2 oz., 12 drs., 11 grs. (Av.).

a, points to the aorta.

term in accordance with the definition given above, is its diminished weight. Dr Church exhibited before the Pathological Society of London the heart of an adult, æt. 47 (who died of cancer of the pylorus), which weighed 3 oz. 1 drachm. The heart, which is represented in figures 255, 256, is still smaller, for it only weighs 2 oz. 12 drs. 11 grs. (avoirdupois); it was removed from the body of a female, æt. 45, the mother of several children, who was under my care for many months, and who died from cancer of the pylorus. When in health she was well nourished, though never fat; her height was 5 ft. 4 in. The case was one of very gradual starvation; I have never seen a more extreme state of emaciation, every particle of fat, and almost all the muscular tissue in the body having disappeared.

In typical cases of cardiac atrophy the heart looks smaller than natural. This is, however, by no means always the case. In some instances, which have come under my own observation, the heart appeared of normal size *when the cavities were relaxed and distended with blood*. It is, I think, possible (though not in simple atrophy from disuse and emaciation such as I am now describing) that an atrophied heart may sometimes be larger than normal in consequence of dilatation of its cavities. I cannot however say that any example has come under my own observation. The general rule is, I think, for the cardiac cavities when relaxed and distended with blood to be of normal size or even less, and for the whole heart when contracted and empty of blood to be smaller than natural.

On microscopical examination, the muscular fibres may present few alterations from the normal. In the extraordinary example represented in figs. 255, 256, the individual fibres are diminished in size (in breadth)—a change which has been described by previous writers—and they contain pigment granules; in short, the microscopical appearances correspond to the condition which has been termed brown atrophy. In some cases of cardiac atrophy, more especially in phthisis and other conditions attended with pyrexia, some of the fibres may be fatty, or in a condition of cloudy swelling.

Symptoms.—In the most perfect examples of atrophy of the heart, such as occur in cancer of the pylorus and phthisis, there are no cardiac symptoms; the heart is still equal to the work which it has to do, and has simply adapted itself, so to speak, to the nature of its surroundings, the atrophy is in fact one of disuse, the volume of blood is diminished, and the patient being precluded from any active exertion, the work of the cardiac pump is reduced to a minimum.

Physical signs.—In well-marked cases, the cardiac impulse is extremely feeble or altogether effaced; the præcordial dulness, both superficial and deep, is much diminished, in extreme cases it may be altogether effaced even during expiration;¹ the cardiac sounds are in some cases normal, but usually weaker than in health. In cases of phthisis the pulmonary second sound is usually more distinct than the aortic, it may be accentuated. The radial pulse is small and weak; in phthisical cases its frequency is increased; in those cases in which the atrophy is due to chronic wasting disease unaccompanied by fever, it may be slower than normal.

Diagnosis.—The positive diagnosis of an atrophied heart is attended with considerable difficulty; for a weak heart which is extensively overlapped by lung cannot be distinguished from a heart which is atrophied.

Atrophy of the heart may be suspected with considerable probability when the patient is much emaciated, when the cardiac impulse is feeble or absent, the præcordial dulness in expiration effaced, the heart sounds feeble, and the pulse small and weak. If, in addition, the observer can satisfy himself that the lungs are not emphysematous (a matter, be it observed, of extreme difficulty in some cases), a positive diagnosis of cardiac atrophy could probably be ventured upon.

¹ In many cases in which the heart is atrophied the lungs are emphysematous. In phthisis the anterior margin of the left lung may of course be consolidated, and in consequence of this fact, and of the over-distention of the right heart which is often present, there may be at least the normal amount of præcordial dulness even when the heart is somewhat atrophied.

Prognosis.—The prognosis entirely depends upon the nature of the primary disease with which the atrophy is associated. The condition of the heart is unimportant, for if we could cure the cancer of the pylorus, the phthisis or other primary affection, the heart would readapt itself to its surroundings, and the atrophy would disappear. (This statement does not of course apply to those cases in which the atrophy is associated with structural lesions of the cardiac walls, such as fibroid degeneration; the prognosis of cases of this nature is considered in other parts of this work.)

Treatment.—Atrophy of the heart due to disuse, and associated with general emaciation, does not call for any special treatment. Our therapeutic measures must be entirely directed to the primary lesion, and to improving the general state of nutrition.

FATTY HEART.

Under the term fatty heart, two distinct pathological conditions are included. In the one—*fatty infiltration* or *fatty overgrowth*—the fatty cells, which are normally present in the sub-pericardial connective tissue, increase in numbers, and the cells of the connective tissue of the myocardium become inflated with oil, so that fatty cells are found in the substance of the myocardium between the muscular elements. In the other, which is termed *fatty degeneration*, oil is deposited within the muscular fibres themselves in the form of very minute (microscopical) molecules and globules. The two conditions (fatty infiltration and fatty degeneration) are not unfrequently combined, but since they are distinct processes, and are often met with independently, they must be considered separately.

FATTY INFILTRATION OR FATTY OVERGROWTH.

Definition.—An excessive development of the sub-pericardial fat, with, in many cases, the development of fat cells between the muscular fibres of the myocardium.

Ætiology.—With rare exceptions fatty infiltration is part and parcel of a general condition in which the fat cells through the body undergo excessive development. All the conditions, therefore, which produce general obesity, are causes of fatty infiltration of the heart. In some persons and in some families, the tendency to excessive formation of fat is hereditary; more frequently the condition is acquired and is due to rich feeding, especially over-indulgence in fat-forming foods, such as fatty, saccharine, and starchy articles of diet, and in alcoholics, sweet ale, porter, rich sweet wines in particular. Inactivity and a sedentary mode of life are also important causes of the condition. Fatty infiltration of the heart is more common in men than in women, and is more frequently met with in the upper ranks of society than in the lower classes.

Pathology and pathological physiology.—In all well nourished individuals some fat is present on the exterior of the heart. Its amount varies very greatly in different cases, and it is only when it becomes distinctly excessive that it can be properly called a disease. Fat cells are found in the sub-pericardial connective tissue, and are collected more particularly in the furrows and depressions on the surface of the heart. Even at time of birth, fat cells are present in the auriculo-ventricular grooves and around the base of the great vessels; they form an elastic cushion or pad which adapts itself to the ever-varying movements of the adjacent parts. In well nourished adults, and more particularly in fat persons, the sub-pericardial fat undergoes considerable increase, and deposits of fat are seen on the surface of the heart, more especially on the anterior surface of the right ventricle. When this sub-pericardial fat is in considerable excess, *i.e.* in cases to which the term fatty overgrowth may be correctly applied, large masses of fat fill the grooves and furrows and a thick layer of fat covers the surface of the organ; the anterior surface of the right heart is first and most affected, but in advanced cases, the whole heart may be encased in a fatty covering. This appearance is well seen in the preparation represented in fig. 260; in it a layer of fat, at least half an inch thick, is situated

on the exterior of the right ventricle. When the cardiac fat is in great excess, it is not confined to the surface of the heart, but is found in the fibrous septa between the muscular fibres of the myocardium (see fig. 257); in some cases, the whole

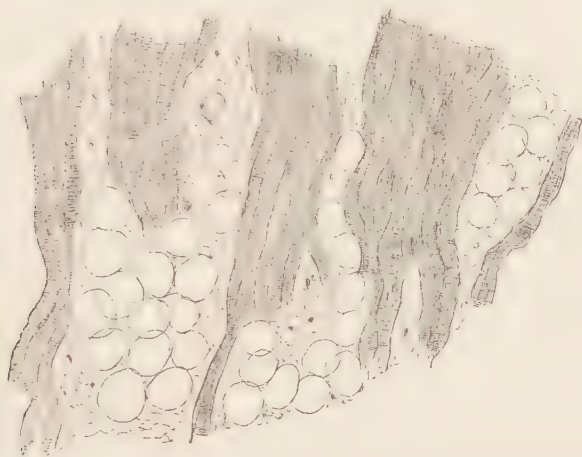


FIG. 257.—*Fatty infiltration of the heart from a section through the wall of the right auricle.*

thickness of the myocardium is invaded, and it is said that the papillary muscles are sometimes implicated. The fat cells encroach, as it were, upon the normal territory of the myocardium, and, in consequence of the pressure which they exercise, may produce atrophy and degeneration of the muscular elements. In advanced cases of fatty infiltration, the much more serious condition, fatty degeneration, is generally present; in some cases, more especially those in which it is limited to special parts of the heart, the fatty degeneration is due to imperfect blood supply, the result of disease of the coronary arteries. This is not in the least surprising, when we remember how much more frequently fatty overgrowth is found in old than in young people; the cardiac and arterial changes are, in fact, in many cases, the common result of a general structural decay.

The effects of fatty overgrowth vary with its extent, and more especially with the condition of the cardiac muscle.

Symptoms.—In slight degrees of fatty infiltration, there are no symptoms nor signs suggestive of cardiac disease. Stout people, whose hearts are covered with an excess of fat, are less capable of active exertion than thin people, and are more or less 'short of wind,' they bear any excessive strain or acute illness badly; but many stout people whose hearts are doubtless covered with an excess of fat enjoy good health and lead active lives. It is only, however, when the myocardium is invaded and the muscular substance of the heart becomes atrophied or degenerated, that symptoms of defective and embarrassed circulation arise.

In advanced cases of fatty infiltration, the symptoms are identical with those of fatty degeneration, which will be presently considered.

Physical signs.—Slight degrees of fatty overgrowth do not produce any perceptible alteration in the physical condition of the heart. In advanced cases the cardiac impulse is feeble, it may be quite imperceptible when the patient is lying on his back, but it can usually be felt when he leans forward or turns on to his left side. In some cases, the apex beat and cardiac impulse, are entirely absent.

The percussion dulness is, as a rule, somewhat increased, but this point is often very difficult to verify for the excessive quantity of fat, both on the outside of the pericardial sac, *i.e.* in the anterior mediastinum, and in the subcutaneous tissue of the chest wall, makes it extremely difficult to define the exact limits of the heart. (The excess of mediastinal and subcutaneous fat is also in part the cause of the diminished impulse and feeble sounds.)

The cardiac sounds are faint and indistinct; in some cases almost imperceptible. In those cases in which the fatty overgrowth is associated with other cardiac lesions, such, for instance, as valvular disease, the character of the heart sounds will, of course, be modified.

In the slighter degrees of fatty overgrowth, the pulse is of good volume and strength, in fact quite normal. In advanced cases, it is small, weak, and sometimes irregular; its frequency

varies, in some cases it is quicker, in others decidedly slower than in health.

Diagnosis.—The diagnosis of fatty overgrowth is often difficult, sometimes impossible. In fat persons we may, even in the absence of any symptoms and signs of cardiac derangement, with much probability suspect, or even with some confidence predict the presence of an increased quantity of sub-pericardial fat, for we know as the result of pathological experience that a considerable increase of the subcutaneous fat is very generally, if not invariably, attended with an increased deposit of fat on the exterior of the heart.

In advanced stages of fatty infiltration, when the action of the heart is weak, we may positively diagnose the condition when the patient is obese, and when we can satisfy ourselves that no other cause for the cardiac weakness is present. In very fat persons it is difficult (as I have mentioned under the physical signs) to ascertain with exactitude the size and strength of the heart by palpating, percussing, and auscultating the præcordial region. The condition of the radial pulse is, in such circumstances, the best guide on which we can rely.

Fatty infiltration in its more advanced stages cannot be distinguished from fatty degeneration; indeed, as I have already pointed out, the two conditions are usually combined.

Prognosis.—A slight excess of sub-pericardial fat is of no consequence whatever. A large excess embarrasses the action of the heart, but provided that it is not attended with symptoms and signs of cardiac weakness, *i.e.* provided that the myocardium is not infiltrated, and that the muscular fibres are not degenerated, it is not necessarily a serious condition. It must, however, be remembered that a heart which is loaded with fat is heavily handicapped in any acute illness or when any serious strain is thrown upon it.

When the muscular tissue is degenerated (*i.e.* when symptoms and signs of cardiac weakness are present) the prognosis

is much more unfavourable, and is identical with that of fatty degeneration. Sudden death may occur from syncope or rupture of the heart. When fatty infiltration is associated with atheroma of the superficial arteries, and presumably therefore with disease of the coronary arteries, the prognosis is also unfavourable.

Treatment.—The objects of treatment are to limit the formation of fat, to promote the absorption of the fatty deposit on the heart, and to invigorate the cardiac muscle. The general health must be kept in the highest possible state of efficiency, the diet strictly regulated, starchy and fatty foods being so far as is compatible with the maintenance of good health omitted from the dietary altogether, sweet ale, porter, and sweet wines should be prohibited; if any stimulant is required a little claret or whisky and water may be allowed. As much active outdoor exercise, as is possible, short of producing fatigue or dyspnœa, should be recommended; walking exercise in a dry, moderately cold and invigorating climate is best. Hill climbing or anything, such as hurrying for trains, which throws a sudden strain on the heart, is to be strictly forbidden. The bowels must be kept regular, and straining at stool avoided, by the administration of suitable purgatives. Liquor potassæ (ten drop doses three times a day) may be given to fat persons with the object of reducing the obese condition. Arsenic is in many cases highly beneficial. When symptoms and signs of cardiac weakness arise, the case must be treated in accordance with the rules which will presently be laid down for the treatment of fatty degeneration.

FATTY DEGENERATION.

Definition.—A degeneration of the muscular fibres of the heart in which the albuminoid constituents of the fibre are split up and microscopical particles of fat are deposited within the muscular fibres; ultimately the transverse striæ disappear, and the functional activity of those muscular fibres, which are affected, becomes completely destroyed.

Ætiology and Pathology.—The muscular fibres of the heart are very liable to be attacked by fatty degeneration, and the condition is met with in a great variety of affections. All conditions which interfere with the supply of oxygen to the muscular tissue, and which seriously derange its nutrition, seem to produce fatty degeneration. Some of these conditions are general; others local. In chlorosis, progressive pernicious anæmia, and, in fact, in all conditions of profound anæmia, fatty degeneration of the heart is developed. It is common as the result of local defects in the blood supply, and is frequently due to atheroma of the coronary arteries. It also occurs in the later stages of those conditions in which the removal of waste products from the cardiac walls is interfered with—in the terminal stages of mitral lesions for example. It is met with in a very intense form in phosphorus poisoning; it results from poisoning by arsenic—a curious fact when it is remembered that arsenic is the best remedy for fatty heart. It is often associated, as we have previously seen, with fatty infiltration. It is not uncommon as the result of acute pericarditis and acute myocarditis, and according to some authorities it is also of frequent occurrence in fibroid degeneration of the heart. (My experience is opposed to this statement, and agrees with that of Dr Charlewood Turner, who failed to find fatty degeneration in several typical cases of fibroid disease. In the very chronic forms of fibroid degeneration the muscular fibres disappear by a process of atrophy rather than by fatty degeneration. In the acute and sub-acute forms of myocarditis fatty degeneration is often, I think, present.) Hypertrophied and dilated hearts are very liable to be affected by fatty degeneration. The condition occurs as the result of long continued pyrexia, and is also met with in many chronic cachectic conditions, such as prolonged suppuration, tubercular disease, cancer, etc. Fatty degeneration is not, however, a constant condition even in severe and typical cases of this sort. In the extraordinary example of atrophy of the heart, shown in figs. 255 and 256, there is no fatty degeneration, so far as I have been able to ascertain.

Fatty degeneration of the heart may occur at any age and in either sex. The anæmic form is more common in women than in men, and in young than in old people. (Chlorosis, which is the most common cause of this form of fatty degeneration, is of course a disease of the female sex and of young women. Progressive pernicious anæmia is also a disease of early and middle adult life, and is, according to some observers, somewhat more common in women than in men.)

The idiopathic form of fatty degeneration, as it is sometimes called (*i.e.* that form which is so frequently associated with disease of the coronary arteries), is more common in males than in females, and is essentially a disease of later life. It has sometimes been called the senile form of fatty degeneration. The form of fatty degeneration which is combined with fatty infiltration is sometimes also called idiopathic.

A heart which is affected with fatty degeneration is, as a rule, somewhat larger than normal. In those cases in which the fatty change attacks a heart which was previously hypertrophied and dilated, the increase in size may of course be great; when the fatty degeneration is the sole cardiac lesion the increase is seldom considerable, and is, for the most part, only apparent, *i.e.* due to the flabby and relaxed condition in which the organ is found after death. In cases of this description the heart is, as a rule, somewhat dilated; in some cases, as in chlorosis and progressive pernicious anæmia for example, some hypertrophy is also present; should the fatty degeneration follow upon long continued suppuration, tubercular disease, etc., the organ may be somewhat atrophied.

Its *colour* is paler than normal, usually of a fawn yellow or pale buff; in some cases, notably in pernicious anæmia, the fatty change is irregularly distributed amongst the muscular fibres; the interior of the heart appears to be dotted over or speckled with little yellow points, which give it a mottled appearance, not unlike the breast of a thrush, to which it has been compared.

The *consistency* is softer than normal, and in advanced

cases the wall of the heart can be readily broken down by the finger. The left ventricle is the part of the heart which is most liable to be attacked, the papillary muscles being in many cases profoundly affected; the right ventricle is the next part to suffer, then the left auricle, and last of all the right auricle.

When the lesion is due to disease of the coronary artery, the fatty change is usually more localised, and may be limited to small portions of the organ, the exact position depends of course upon the distribution of the branch of the artery which happens to be affected.

On microscopical examination, the affected muscular fibres are seen to contain little molecules of oil which stain black with perosmic acid; these molecules are very minute, and are, for the most part, singularly uniform in size; in some cases they coalesce and form minute globules which seldom, however, exceed the size of half a red blood corpuscle. The fatty molecules are sometimes arranged in rows, but are usually distributed in an irregular manner throughout the whole thickness of the fibre; as the change progresses, the transverse striæ become indistinct and finally disappear, the whole fibre ultimately becoming filled with little oily particles. (See fig. 258.)

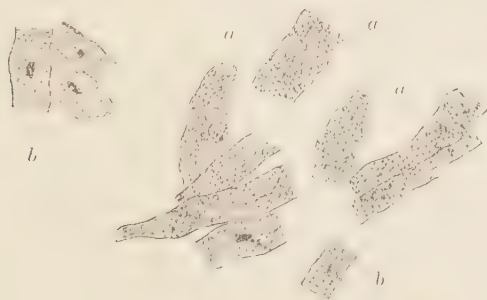


FIG. 258.—*Muscular fibres of the heart in a case of fatty degeneration.*

a, a, a, fibres in which the fatty change is just commencing; *b, b*, fibres in which it is far advanced.

S S

Muscular fibres affected with fatty degeneration are much more brittle than healthy ones, and readily break up transversely into short fragments.

In fatty hearts it is not at all uncommon to find the condition which has been termed brown atrophy, in which pigment particles are deposited around, more particularly at the ends or poles of the muscle nuclei. The connective tissue nuclei, and the fibrous septa between the fibres are sometimes also increased; this change is chiefly, I think, observed in hypertrophied hearts, more especially in those conditions, such as mitral regurgitation, in which the venous return from the cardiac walls is interfered with. Fat cells are, of course, seen between the muscular fibres, in those cases in which fatty degeneration is combined with fatty infiltration.

The functional activity of a muscular fibre, which is affected with even a moderate degree of fatty degeneration, is seriously interfered with; in the final stages of the process the fibre loses its contractility altogether. In those cases, therefore, in which a large number of the muscular fibres are affected, and in which the change is widely distributed throughout the heart, the cardiac pump is of necessity greatly weakened. Under such circumstances we should of course expect the systemic arterial system to be under distended and the systemic venous system to be over distended with blood; and in chlorosis and progressive pernicious anæmia, in which affections the fatty change reaches a high degree of intensity and is widely distributed throughout the heart, these results do actually occur. In these affections, too, the heart becomes both dilated and hypertrophied, and relative and muscular incompetence at the mitral and tricuspid orifices are not unfrequently established.

When the fatty degeneration is combined with fatty overgrowth independently of the anæmic condition, the same sequence of events is often observed.

In some cases of fatty degeneration, arterial anæmia is the only result; and dilatation of the ventricles, regurgitation at the mitral and tricuspid orifices, and engorgement of the

systemic veins, are not observed. These differences are sometimes very difficult to explain. In some cases the absence of cardiac dilatation and venous engorgement is probably due to the fact that the fatty change, although reaching a high degree of intensity in certain fibres, is limited in distribution, and the healthy fibres, which remain, are able to carry on the work of the circulation provided that the organ is not called upon to make any sudden effort. In others, the explanation is probably to be found in the fact that the patients are old people in whom the tissue changes are at a minimum, and who lead tranquil, placid lives. Such patients suffer little so long as they keep quiet, the slightest extra exertion, however, brings a shortness of breath; if they continue to exert themselves, or, if in consequence of deranged cardiac and vaso-motor innervation, or any other cause independently of muscular exertion, the heart has continuously to exceed the quiet minimum of work, so to speak, of which it is capable, the other secondary consequences (dilatation, venous engorgement, auriculo-ventricular regurgitation) would doubtless follow. In others again, the absence of symptoms and signs of venous engorgement is probably due to the fact that the mitral orifice is unyielding owing to senile (atheromatous) changes in the mitral ring, in consequence of which relative incompetence of the mitral orifice does not occur.

The extent, and more particularly the distribution, of the fatty change, the amount of work which the heart is called upon to do, the state of the mitral ring, and the condition of the cardiac and vaso-motor nerve mechanisms seem to me to be the chief factors to which we must look in order to explain the different results which are met with in these two classes of cases. That the condition of the central nervous system exerts a most important influence upon the secondary results of cardiac disease is, I think, perfectly clear. I have seen, for example, a case in which the arterial tension was high, the left ventricle hypertrophied and dilated, the mitral valve incompetent and somewhat contracted the result of old rheumatic disease, the lungs in a condition of brown atrophy, the

right ventricle hypertrophied and considerably dilated, and in which a very considerable amount of fatty degeneration of the hypertrophied right and left ventricles was also present, in which there was extreme shortness of breath on exertion, and yet in which there was never any subcutaneous dropsy. Such a result can only, I think, be explained by supposing that the condition of the blood and of the peripheral vessels, probably in consequence of some peculiarity in the nerve or nervo-muscular tone, prevented the occurrence of subcutaneous effusion.

Symptoms and physical signs.—After what I have just stated, it will be at once understood that there are several types of fatty heart, and that the symptoms and physical signs differ very materially in different cases.

The symptoms and physical signs of the fatty degeneration of the heart, which is due to general anæmia, and which is met with in its most typical form in chlorosis and progressive pernicious anæmia, have been previously described in treating of mitral regurgitation. (See page 441.)

When the fatty degeneration is combined with fatty infiltration, the subcutaneous fat is almost invariably well developed, all degrees of obesity being met with ; the colour of the skin is usually pale, but should mitral or tricuspid incompetence be at the same time present, blueness of the lips and the other external manifestations of peripheral venous engorgement may of course be present. In cases of combined fatty degeneration and fatty infiltration, the skin often has a greasy, unctuous feel, and this is more especially the case when the patient is intemperate.

In other cases, more especially, I think, in those in which the fatty degeneration depends upon disease of the coronary arteries, the patient is rather the reverse of fat ; the peripheral arteries stand out prominently, and the arcus senilis is often present. The abdomen is not unfrequently covered with a considerable layer of fat, and the big, pendulous belly contrasts forcibly with the flabby and, comparatively speaking, attenuated extremities. (General atheroma, disease of the

coronary arteries, and the arcus senilis, are of course frequently observed in *fat* people whose hearts are fatty.)

The subjects of fatty degeneration of the heart are, as a rule, incapable of concentrated mental effort and of any active bodily exertion; this is more particularly the case when the fatty change is widely distributed throughout the heart. Cases are, however, not uncommon, in which persons who have been leading active and useful lives suddenly die from fatty heart. In these cases the fatty change is usually limited in distribution. I have known three instances in which professional men—two physicians and one clergyman—have suddenly died from rupture of the heart, all of them having been actively engaged until within a day or two of death in professional work. In all of these cases the coronary arteries were atheromatous, and the fatal rupture was apparently due to arrested blood supply and acute localised softening.

A ‘sinking’ sensation is sometimes complained of in the pit of the epigastrium; in some cases there is actual pain in the region of the heart; occasionally true attacks of angina pectoris occur.

Shortness of breath on exertion is usually a prominent symptom, and exertion often causes a dry, irritable, cough. In advanced cases, the dyspnœa may become constant. ‘Cheyne-Stokes’ respiration sometimes precedes the fatal issue.

Some of the more prominent symptoms are due to anæmia of the brain. The memory is, in many cases, impaired; the patient loses his former decision of character and ‘nerve,’ and may become wayward and irritable. Vertigo is a common symptom; and in advanced cases, fainting is not unfrequently observed, getting up from the recumbent to the sitting or standing position, or raising the head suddenly, may bring on an attack of syncope. Pseudo-apoplectic attacks, in which the patient remains unconscious for some time, are met with in some cases. When fatty degeneration is combined with other cardiac lesions, such, for example, as mitral regurgitation, other symptoms, which I need not here detail, are of course present.

heart. The cardiac impulse is feeble, and in some cases, more especially in those in which the chest wall is covered by a thick layer of fat, altogether effaced. The impulse, when perceptible, is more diffused than in health. The præcordial dulness may be quite normal or slightly increased; in those cases in which the heart is dilated or hypertrophied from associated disease the increase may, of course, be considerable. In typical cases, and more especially when the heart is acting quietly, the first sound is feeble, muffled, and distant; its duration is considerably shorter than in health. These characters are not, however, observed in all cases. In chlorosis, for example, the heart is very irritable and easily excited, and the first sound, although shorter than in health, is usually quite distinct, its tone is raised, in fact it may present all the characters which have been described under the head of cardiac dilatation. The same character of the first sound may be present in other forms of fatty degeneration when combined with dilatation. Mitral and tricuspid systolic murmurs are of frequent occurrence, more especially in anæmic cases.

The characters of the pulse differ very considerably in different cases, and depend upon the irritability of the cardiac muscle, the presence or absence of dilatation, and the condition of the valvular apparatus.

In some cases (more especially in anæmic cases and in those in which the fatty degeneration is combined with dilatation) the pulse is quicker than normal; in others (more particularly, I think, when the fatty degeneration is associated with general atheroma and disease of the coronary arteries) the pulse frequency is diminished.

In advanced cases the rhythm is often irregular, though exceptions to this general statement frequently occur. When the fatty degeneration is combined with dilatation and mitral disease, the pulse may be extremely irregular, and some of the pulse waves may fail to reach the wrist. In typical cases of fatty degeneration, the pulse is small and extremely weak—an exception occurs in chlorotic cases, in the earlier stages of which the arterial tension is increased.

Physical signs.—The physical signs are those of a weak

Diagnosis.—The diagnosis of fatty degeneration of the heart is, in some cases, attended with great difficulties. In order to come to a positive conclusion we must, in the *first* place, satisfy ourselves that the action of the heart is permanently weak; and, in the *second* place, that there is no other cause for the cardiac weakness (such as fibroid degeneration, mitral disease, etc.).

Too much importance should not be attached to the results of the physical examination; for, on the one hand, it is extremely difficult to determine the exact physical condition of the organ in those cases in which the chest wall is thickly covered by subcutaneous fat, or in which the heart is overlapped by emphysematous lungs; and, on the other, the agitation, which many patients experience when they come to consult a physician, induces overaction of the heart, increases the intensity of the cardiac sounds and the frequency of the pulse, and often leads to the belief that the organ is stronger than it actually is. It is very essential, therefore, in all cases, in which the symptoms are suggestive of fatty heart, and particularly in those cases in which there is reason to suppose that the action of the organ is modified by nervous causes, to examine the patient more than once, and, as Professor Gairdner has forcibly pointed out,¹ under various conditions of cardiac action, before committing ourselves to a positive opinion. Feeble cardiac impulse, a short and muffled first sound, a weak pulse, and symptoms of cerebral anæmia, are the positive facts which are of most importance, and which enable us to make the *first step* in the diagnosis, viz., that the heart is weak.

The *second step*, viz., that the weakness is due to fatty degeneration of the heart, is chiefly attained by the method of exclusion. We can only give a positive diagnosis of fatty heart when we are satisfied that the cardiac weakness is not a mere temporary condition, and when we have excluded valvular lesions (more particularly mitral valve lesions), cardiac dilatation, myocarditis, fibroid degeneration, and adherent

¹ Russell Reynolds' *System of Medicine*, vol iv. p. 546.

pericardium, in all of which conditions the same indications of cardiac weakness may be present.

Fatty degeneration may of course be combined with any of these lesions, and in many cases in which these conditions (mitral lesions, cardiac dilatation, etc.) are present, we may, from the progress of the case and from the nature of the symptoms and physical signs, have good reason to *suspect* that the cardiac muscle is fatty. Mere suspicion or probability does not, however, justify a positive diagnosis.

The absence of any marked symptoms and signs of pulmonary and systemic venous engorgement, the absence of endocardial murmurs, the fact that the cardiac muscle does not respond well to cardiac tonics (*i.e.* to digitalis), the general condition of the patient (corpulence, an atheromatous condition of the superficial arteries, the presence of an arcus senilis, a history of intemperance) are all strongly in favour of fatty heart, and enable us to exclude most of the other causes of cardiac weakness and defective circulation which I have just mentioned.

Mitral lesions sufficiently severe to produce the symptoms which are present in typical cases of fatty degeneration, such as we are now considering, would in all probability be attended with dropsy and other symptoms and signs of venous engorgement—mitral murmurs would probably be present, and there would be distinct evidence of enlargement of the right heart. In advanced stages of mitral regurgitation the systolic apex murmur sometimes vanishes, but can almost invariably be re-established by the administration of digitalis; in advanced cases of mitral constriction the murmur is very often, usually indeed, absent. Now, in pure cases of fatty degeneration (*i.e.* cases uncombined with any considerable amount of dilatation or with mitral or tricuspid regurgitation) there are no symptoms of venous engorgement, or, at all events, such symptoms are slight. There is little difficulty, therefore, in making a distinction between pure cases of fatty heart and cases of mitral regurgitation.

It is, in most cases, impossible to distinguish fatty degeneration of the heart and chronic myocarditis; fibroid

degeneration sufficiently severe to cause the symptoms and physical signs which characterise advanced cases of fatty degeneration, would be more likely to be attended with considerable dilatation, and with symptoms and signs of venous engorgement.

In women at the meno-pause, attacks of syncope and other indications of defective circulation are not uncommon. Should the patient be stout, it may be impossible to exclude fatty heart. The diagnosis can only be made by watching the future progress of the case.

In chlorosis and other conditions of advanced anæmia the diagnosis of fatty heart can be positively made, for we know as the result of pathological experience, that the cardiac muscle is in a condition of fatty degeneration in these cases.

Prognosis.—The prognosis of fatty degeneration of the heart depends upon the cause. In chlorotic cases the patient will almost certainly get well; in progressive pernicious anæmia the prognosis is very hopeful if arsenic be systematically administered in the manner which I have previously described. The fatty degeneration which attends long continued pyrexia is generally recovered from. The fatty degeneration of old age; that which results from disease of the coronary arteries; and that which attacks hearts affected with chronic valvular disease or other permanent structural lesion (such as the hypertrophy of chronic Bright's disease, the hypertrophy which attends emphysema, etc.), is seldom, if ever, recovered from.

In trying to form a forecast of the probable course of the case, it is important to remember that fatty degeneration is a frequent cause of sudden death; the fatal result may be due to syncope, rupture of the heart, or angina pectoris.

Fatty degeneration, however slight, adds very seriously to the dangers of an acute illness. Persons whose hearts are fatty bear pain badly, and are unfavourable subjects for severe operations. Should they require an anæsthetic, ether, or a mixture of ether and chloroform, should be administered to them. (But they are not, in my opinion, exceptional in this

respect, for, so far as I can weigh the experimental and other evidence, ether is a much safer anæsthetic than chloroform ; the practical advantages which attend the administration of chloroform do not, so far as I can judge, at all counterbalance the greater risks to which it subjects the patient.)

Treatment.—The treatment of fatty degeneration of the heart varies somewhat in accordance with the cause. Our main object is, of course, to remove the condition.

In anæmic cases, iron, arsenic, or a combination of these drugs, must be given, and the general treatment, which has been previously described in treating of mitral regurgitation (see p. 463), carried out. Great care must be taken in these, and indeed in all cases of fatty or weak heart, to avoid throwing any sudden strain upon the damaged organ, but this point will be again referred to presently.

In the fatty degeneration which follows a severe attack of typhus or other continued fever, the treatment must be essentially tonic ; quinine, iron, and strychnine are useful drugs. When the cardiac weakness is extreme the patient must be cautioned against making any sudden effort or doing anything which is likely to induce cardiac syncope. The dietetic and other measures suitable for a convalescent—and which it is unnecessary to mention here—must of course be prescribed. Cardiac tonics and stimulants (digitalis, brandy, ammonia, etc.), are required when the cardiac weakness is very great ; and since the fatty change may be established before the period of convalescence is reached, the remedies may of course be required during the febrile stage of the disease.

The treatment of the fatty degeneration which is so often established in the terminal stages of cardiac valvular lesions, hypertrophy of the heart, etc., has already been considered under the different valvular lesions.

In treating the senile form of fatty degeneration, we can seldom, if ever, expect to remove the cause, and our treatment must therefore be for the most part palliative. All sources of cardiac strain, or anything likely to induce cardiac syncope, must be carefully guarded against. All sudden efforts must

be forbidden ; all sources of mental worry, anxiety, or excitement avoided ; in fact, the patient must, so far as is possible, considering his mental disposition and surroundings, be made to lead a quiet and regular life. The patient should be cautioned against suddenly rising from the recumbent position ; warm baths and powerful purgatives are not permissible in those cases in which there is a tendency to syncope, and in which symptoms of cerebral anæmia are prominent. The general health must be maintained in the best possible state of efficiency. The diet must be digestible, but at the same time nutritious, and the quantity of food taken at each meal should be strictly moderate. Flatulent distention of the stomach should, if possible, be prevented, for a distended stomach pushes up the diaphragm and seriously impedes the action of the heart. A heavy meal should on no account be taken just before going to bed, or the patient will be likely to wake up with troublesome dyspnœa, or perhaps alarming symptoms of cardiac failure. I have known more than one case in which a patient who had taken a hearty supper awoke during the night and suddenly expired, and in which the exciting cause of the attack seemed to be overloading of the stomach. The bowels must be kept regular, and the condition of the skin carefully attended to.

Plenty of fresh air is eminently desirable ; the proper ventilation of the rooms in which the patient lives during the day and sleeps during the night, is a very important point. As much outdoor exercise as is possible, short of producing fatigue or dyspnœa, is desirable. Unfortunately in advanced cases, even gentle exercise cannot be indulged in. Should symptoms of sudden cardiac failure arise, brandy, ammonia, ether, or other stimulant remedies, should be promptly administered. Attacks of dyspnœa, cardiac pain, angina pectoris, etc., must of course be met as they arise, by appropriate remedies. Arsenic, iron, and strychnine are the most useful drugs. In some cases, more especially where there is associated mitral valvular disease or cardiac dilatation, digitalis is of the greatest service ; but in others it seems useless or even prejudicial, it increases the arterial tension, and so

throws an increased strain upon the heart, without (in some cases) appearing to produce any beneficial influence upon the damaged cardiac wall.

SPONTANEOUS RUPTURE OF THE HEART,

Ætiology and Pathology.—Spontaneous rupture of the heart probably never occurs when the organ is perfectly healthy, but is always the result of disease. It is occasionally, though rarely, due to acute softening of the cardiac wall, the result of acute local myocarditis or thrombosis of the coronary artery; in most cases it is due to chronic softening, the result of fatty changes, both fatty infiltration and fatty degeneration, but especially the latter. The bursting of an aneurism of the cardiac wall is another, but a very rare, cause of the condition.

In the majority of cases it is the wall of the left ventricle which gives way, doubtless in consequence of the facts that the degenerative changes, which are the causes of the rupture, more frequently involve the left ventricle than the other parts of the heart, and that the blood pressure within the cavity of the left ventricle during its systole is much greater than in the other cavities of the organ. The rupture is usually situated in the anterior wall of the ventricle in the neighbourhood of the septum; in some cases it is placed in the posterior wall; exceptionally it is the wall of the right ventricle which gives way. The burst sometimes occurs when the patient is making a sudden effort, such as hurrying for a train, or strain at stool; in other cases, it takes place while he is at perfect rest.

The size of the rupture varies from a mere slit to an extensive lacerated opening; in some cases, the external opening (*i.e.* the opening into the pericardium) is smaller than the internal.

The effect of the rupture is, of course, to allow the escape of blood from the cavity of the heart into the sac of the pericardium. When the laceration is extensive and a large quantity of blood escapes, death may be instantaneous; in other cases, more especially when the rupture is a small one, and the direction of the rupture oblique, the patient may

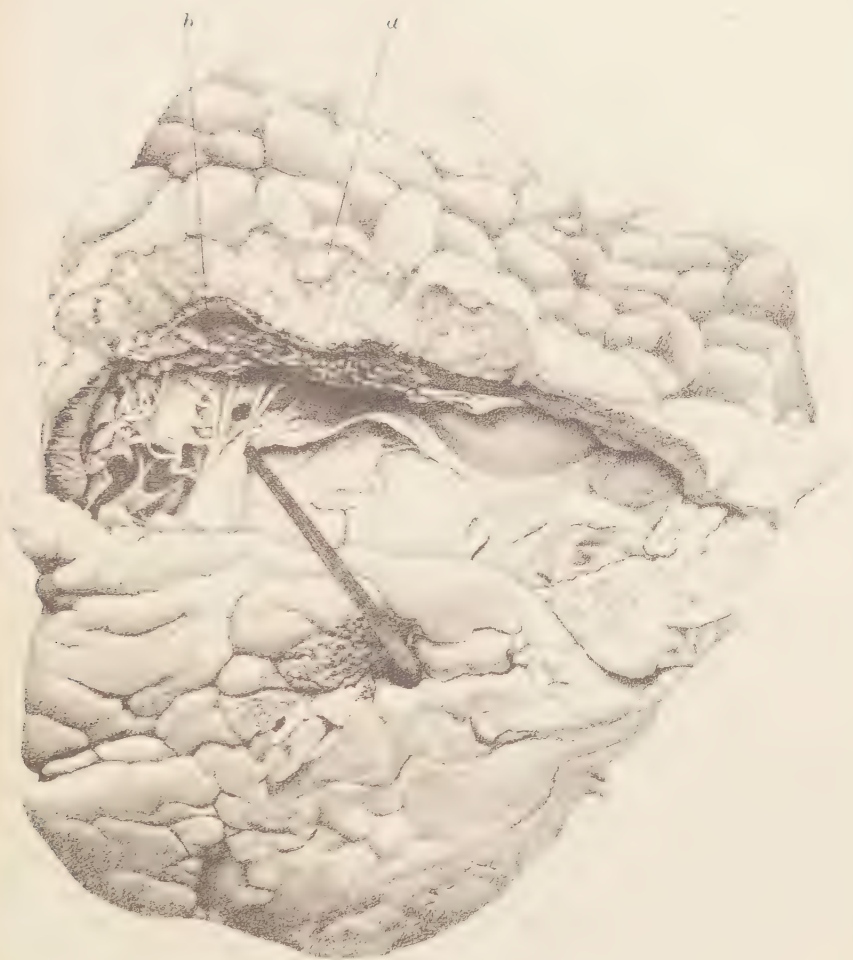


FIG. 259. *Rupture of the Left Ventricle seen from the outside. (Natural size.)*

The heart is covered by a thick layer of fat, which at the point *a* is five-eighths of an inch in thickness; *b*, muscular substance beneath the external layer of fat. A piece of whalebone has been placed in the external orifice of the rupture.

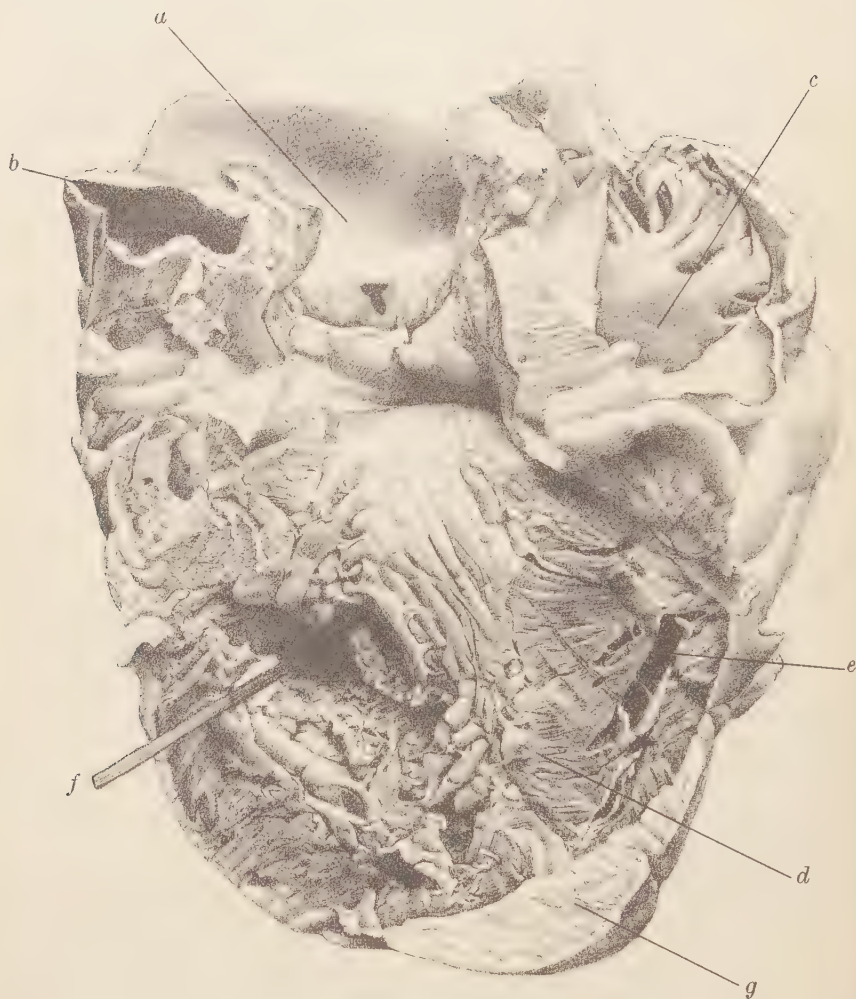


FIG. 260. *Rupture of the Left Ventricle. (Natural size.)*

The heart has been so cut open that the septum ventriculorum and the interior of both ventricles are seen. A piece of whalebone is inserted into the rupture.

a, interior of the aorta; *b*, pulmonary artery transversely divided; *c*, interior of right auricle; *d*, septum ventriculorum; *e*, interior of right ventricle; *f*, piece of whalebone placed in rupture; *g*, layer of fat on the exterior of the heart.

live for several hours, or even for some days, after the occurrence of the accident.

Symptoms and physical signs.—The occurrence of rupture of the heart is attended with sudden and severe pain in the heart; if the patient is standing or walking when the accident occurs, he will probably fall to the ground. In those cases in which death does not immediately occur, there are all the appearances of collapse, and in addition to the pericardial pain and oppression, the patient complains of great difficulty in breathing. When the patient survives sufficiently long to be seen by a physician, the pain, dyspnœa, and collapse, are usually so great as to prohibit a minute examination of the chest. The specimen represented in figures 259 and 260 is typical, both as to its pathological characters and the clinical symptoms which were present during life. The history of the case is as follows :—

A. B., a parish patient, æt. 67, a big, stout woman who had previously enjoyed fair health, was suddenly seized one morning on getting out of bed to get a drink, with a severe pain in the region of the heart. Her neighbours, who lived in the room beneath, heard her fall heavily on the floor, and on proceeding to her assistance they found her in a semi-conscious condition. On my arrival shortly afterwards, I found her conscious, but in a condition of profound collapse; skin covered with a cold clammy sweat, pupils dilated, voice husky, pulse hardly perceptible. There was great difficulty in breathing; she had vomited; she complained of intense pain and oppression in the region of the heart; the præcordial region was exquisitely tender to the touch, and I was consequently unable to ascertain the extent of the percussion dulness; the heart sounds were not heard. Morphia and brandy were administered, and a soap and opium liniment was applied over the region of the heart. The patient died eight hours afterwards. On making a *post-mortem* examination I found the pericardial sac distended with black clotted blood. The rupture shown in the drawing was situated in the anterior wall of the left ventricle close to the septum. The internal aperture was ragged, and considerably larger than the external. The heart was in an advanced condition of fatty infiltration, a layer of fat, fully half an inch thick, being situated on the exterior of the right ventricle. At the seat of the rupture the muscular fibres were in an advanced condition of fatty degeneration; the coronary arteries were atheromatous, and the branch supplying the ruptured part of the heart was obstructed.

Diagnosis.—The sudden occurrence of severe pain in the region of the heart, followed by collapse and difficulty of breathing, is suggestive of rupture of the heart. Should the patient survive a sufficient length of time, it may be possible to detect the enlargement of the pericardial sac by means of percussion.

Prognosis.—The prognosis in cases of spontaneous rupture is hopeless. In no case, so far as I know, has recovery taken place.

Treatment.—Little can be done in the way of treatment. Morphia should be given to allay the pain, and a cold anodyne liniment applied to the præcordial region. The profound collapse which, in many cases, threatens to prove immediately fatal, and which indeed in some cases does prove fatal, suggests the administration of stimulants; but it is important to remember that remedies which give increased strength to the heart are injurious, in so far as they favour an increased quantity of blood being expelled from the cardiac cavity into the sac of the pericardium. Stimulants should only, therefore, be administered in those cases in which the collapse threatens to prove fatal.

WAXY DEGENERATION OF THE HEART.

Waxy degeneration of the heart, which is not very uncommon in cases in which the lardaceous change is widely distributed throughout the body, has recently been described by Professor D. J. Hamilton; but since it is not characterised by any distinctive cardiac symptoms or signs, and is of pathological rather than of clinical importance, it is unnecessary to refer to it in detail here.¹

NEW GROWTHS IN THE HEART.

Ætiology and Pathology.—The heart is occasionally the seat of new growths; primary tumours are extremely rare, secondary deposits (more especially cancerous and

¹ *Journal of Anatomy and Physiology*, Oct. 1883, p. 54.

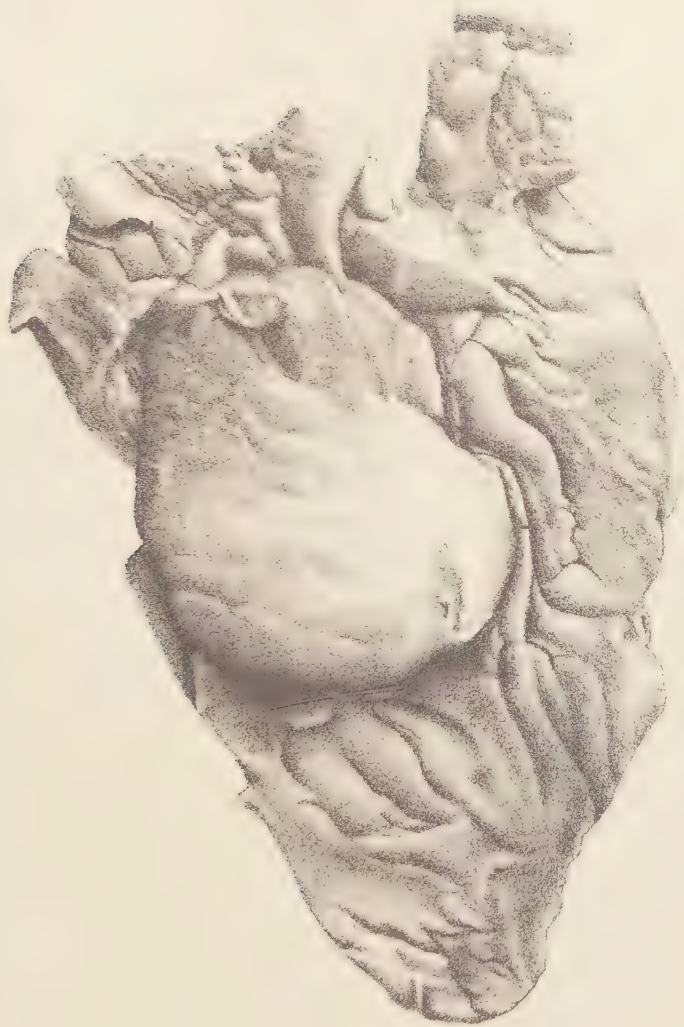


FIG. 261. *Tumour on the exterior of the Heart. (Natural size.)*

The patient died suddenly a few days after delivery; she had never complained of cardiac symptoms.

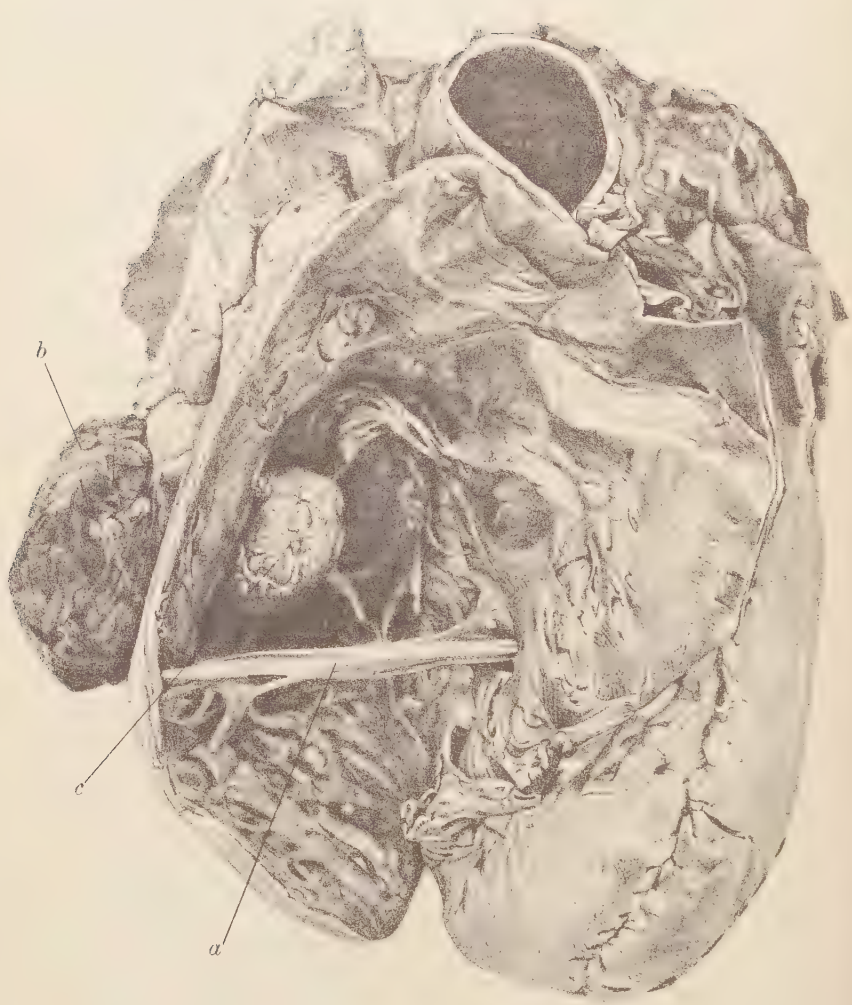


FIG. 262. *Sarcomatous tumours in the heart. (Natural size.)*

The walls of the right ventricle, which have been cut open, are kept apart by a piece of stick (*a*).

b, tumour on the exterior, and *c*, tumour on the interior of the right ventricle; the internal tumour is situated immediately below the tricuspid orifice.

Copied by Professor Turner's permission from a specimen in the Anatomical Museum of the Edinburgh University.

sarcomatous tumours and hydatid cysts) are more common. The more important of the new growths which have been met with in the heart are:—

1. *Cancer*.—Very few instances of primary cancer of the heart are on record; secondary deposits of scirrhus and encephaloid are occasionally met with, and are said to occur more frequently on the right than on the left side of the organ. The cancerous nodules are usually situated on the surface of the organ, and are then sub-pericardial; in some cases they are sub-endocardial; occasionally, though rarely, they are embedded in the myocardium. Pericarditis occurs in some cases in which the cancerous nodules are situated beneath the visceral pericardium, and is still more common when a cancer of the lung or mediastinal tissues makes its way through the parietal layer of the sac.

2. *Sarcomata*.—Various forms of sarcoma (lympho-sarcoma, melanotic sarcoma, etc.) have been met with in the heart, and are probably more common than cancer. They are usually secondary. The individual tumours may attain to considerable size, as in the specimen represented in fig. 261. The sarcomatous nodules may be situated on the surface of the organ, beneath the endocardium, or in the substance of the myocardium. In rare cases, a remarkable example of which has just come under my notice in the *post-mortem* theatre, a malignant tumour of the mediastinum may make its way through the wall of the heart or down the superior vena cava, and form a tumour in the interior of the heart. In the case to which I refer the cavity of the right auricle was almost entirely filled by a large mass of new growth. The sub-pericardial and sub-endocardial forms of cardiac sarcoma are well seen in fig. 262. Sarcomatous tumours on the surface of the heart have not the same tendency to produce pericarditis as cancers.

3. *Tubercle*.—Miliary tubercles are occasionally, though more rarely than might be expected, met with in the tissues of the pericardium, and are always associated with tubercular nodules in other organs; they are much more common in children than in adults; some pericarditis is usually present.

coronary artery.¹ In the specimen represented in fig. 263, which I met with in the *post-mortem* theatre of the Edinburgh Royal Infirmary during the past session (the specimen occurred in a case of Dr Wyllie's, with whose permission it is represented here) a cyst the size of a small orange, containing partly fluid and partly clotted blood, is situated in the anterior wall of the left auricle, *i.e.* between the auricle and the posterior surface of the base of the aorta. The aortic orifice was extremely stenosed, the two cusps (there were only two cusps) being converted into thick, dense, calcareous masses; there had apparently been old ulceration at the base of the cusps, and between them a depression, which passed backwards in the direction of the cyst, existed, and seemed to represent an old ulcer. The coronary arteries which passed round the wall of the cyst were pervious. The cyst was probably I think, an aneurism which had originally communicated with the left ventricle at the point of depression, situated between and just below the two segments of the aortic valve; the orifice had become obliterated, and the sac partly filled up by deposits of laminated fibrine.

7. *Fibroid tumours*.—Vegetations which look like fibroid growths are occasionally met with; they usually spring from the mitral valve-ring, and grow upwards into the cavity of the left auricle. A very beautiful specimen, which seemed to be of this description, came under my observation in the *post-mortem* theatre of the Edinburgh Royal Infirmary a few months ago.

Symptoms and physical signs.—In many cases the presence of the new growth is not manifested either by symptoms or physical signs; in fact, in most cases tumours of the heart are of pathological rather than of clinical interest.

In some cases (more especially in the cancerous and tubercular forms), pericarditis is established. In others (more especially in the case of hydatid tumours projecting into the cardiac cavities, sub-endocardial sarcomata, syphilitic gummata in the ventricular walls near the insertion of the valves,

¹ *Transactions of the Pathological Society of London*, vol. ix. p. 165.

and the rare fibroid growth which springs from the mitral ring) the tumour is so situated as to produce obstruction or to interfere with the perfect closure of one or other of the valvular orifices. In such cases the usual symptoms and signs of valvular disease may be present.

Diagnosis.—Primary tumours in the heart cannot be diagnosed during life. Secondary deposits may be suspected when the symptoms or physical signs of pericarditis or valvular disease arise in the course of a case in which there are (or have been) new growths of a malignant or hydatid character in other parts of the body.

Prognosis.—It is unnecessary to dwell on the prognosis, since it is in most cases impossible to recognise the presence of the new growth during life.

Treatment.—In those cases in which pericarditis or valvular disease is present, the treatment which has been previously recommended for those conditions must be practised. When there is reason to suspect that the cardiac derangement depends on tubercle or syphilis, the remedies which are appropriate for those affections must be administered. When there is reason to suppose that the heart is affected with cancerous or sarcomatous growths, the systematic administration of arsenic should be tried.

CHAPTER VII.

THE CARDIAC NEUROSES. PALPITATION. INTERMITTENT ACTION.
ANGINA PECTORIS.

Cardiac Neuroses.

UNDER the head of cardiac neuroses are included a variety of conditions, the most prominent feature of which is derangement of the nervous mechanism of the heart. In some cases, the motor and co-ordinating part of the mechanism is affected ; in others, the sensory part is implicated ; while in others both the motor and sensory functions are involved.

In many cases, the heart itself is perfectly healthy ; the cardiac affection is then purely functional. In other cases, the neurotic derangement is either caused by, or is associated with, structural lesions in the heart, aorta, or pericardium.

Palpitation, irregular and intermittent action, and cardiac pain, are the most important external manifestations of these neurotic disturbances.

PALPITATION OF THE HEART.

Definition.—Palpitation of the heart is a symptom not a disease. It consists of excessive action of the organ. The frequency of the heart's contractions is usually, but not always, increased ; the cardiac action, which is in some cases irregular or intermittent, is vividly, and sometimes painfully, perceptible to the patient. The condition is essentially paroxysmal.

Ætiology and Pathology.—There are two great forms of palpitation, viz., organic and neurotic or functional.

1. *Organic palpitation*.—In this form the heart is diseased, and the palpitation is usually an indication of cardiac weakness. Whenever, in fact, the heart (whether healthy or diseased) is called upon to make a greater effort than its reserve force is equal to, palpitation is apt to be experienced. Palpitation is consequently a common symptom in cardiac dilatation, and in cases of failing hypertrophy. The exciting cause of the palpitation is very commonly a sudden increase of the peripheral resistance, the result of muscular effort, vasomotor constriction, etc. In other cases, organic palpitation is the result of excessive irritability of the cardiac muscle. This is notably the case in the fatty degeneration of anæmia, in which condition attacks of palpitation are extremely common and are at once produced by any exertion.

2. *Neurotic palpitation*.—In typical cases of neurotic palpitation there is no organic cardiac lesion, but the deranged action is due to disturbance, either direct or reflex, of the motor and co-ordinating mechanism of the heart.

3. In a third group of cases the organic and neurotic forms of palpitation are combined. In anæmia, for example, the cardiac nerves as well as the cardiac muscle are unduly irritable, and palpitation is very readily produced by mental and emotional causes (*i.e.* causes acting through the nervous system) as well as by muscular effort and other conditions which throw an increased strain upon the irritable and weakened heart.

The following description will be limited more especially to the neurotic form of palpitation, though I shall incidentally refer to the organic variety.

Neurotic Palpitation.

Ætiology and Pathology.—Neurotic palpitation is a very common condition, and is of considerable practical importance, for it not only causes discomfort and anxiety at the time of the attack, but persons who are affected by it are very apt to suppose that the heart is organically diseased, and to labour under the fear of sudden death. Neurotic palpitation is frequently observed in healthy persons, more especially in

emotional and excitable individuals. Persons of a phlegmatic disposition, and those who have strong control over themselves, *i.e.* 'cool people,' are not often affected by it when in health.

Pathologically we find that anything which lowers the nerve tone and power of self-control predisposes to this form of palpitation. It is apt to occur during convalescence from severe disease; it is met with in all forms of anæmia; sedentary occupations, unhealthy surroundings, insufficient food, bad ventilation, want of sun-light, are therefore powerful predisposing causes; it is particularly apt to arise in consequence of sexual excesses, mental worry and anxiety, over work and loss of sleep. Prolonged muscular effort, more especially when combined with great mental excitement and insufficient food, sometimes gives rise to it—witness the frequency of 'irritable heart' and palpitation in the soldiers who took part in the great American War. Palpitation is very common in hysteria; is frequently caused by over indulgence in tea and tobacco; is frequently seen in connection with dyspepsia and gout; is a very prominent symptom in exophthalmic goitre (Grave's disease); and is sometimes produced reflexly by uterine and ovarian derangements, worms in the intestine, etc.; it is too of common occurrence in the early stage of pulmonary phthisis.

Neurotic palpitation is much more common in early adult life than at any other period; and is more common in women than in men, though very severe palpitation is frequently met with in young males, usually, I believe, as a result of sexual excesses and over indulgence in tobacco. The exact manner in which the nervous mechanism is deranged cannot always be ascertained. Theoretically we may suppose that palpitation may be due to the following conditions:—

1. Excessive stimulation or over irritability of the ganglia and nerves in the heart itself. This is probably, in part at least, the cause of the palpitation of anæmia, and of that due to tea and tobacco.

2. Excessive action of the sympathetic—the cardiac accelerator.

3. Defective action of the vagus—the inhibitory nerve of the heart.

In exophthalmic goitre there is probably an organic lesion of the cervical sympathetic; in other cases, as for example, in locomotor ataxia, in which attacks of palpitation analogous to the so called *gastric crises* are sometimes observed, the lesion (which produces the palpitation) is probably situated in the cervical portion of the spinal cord, or in the medulla oblongata. In other cases, as for example in hysteria, the higher (cerebral) centres are at fault; while in others, the derangement is reflex and due to peripheral irritation.

The exciting cause of neurotic palpitation is very generally a sudden start or other form of emotional disturbance. The palpitation is usually aggravated, and is sometimes produced when the patient's attention is directed to the heart. Neurotic palpitation is rarely followed by any permanent cardiac lesion. In one case, which has come under my observation, in which a young man was suddenly attacked during the night with most violent palpitation, slight aortic regurgitation subsequently developed. I have always suspected that in that case one of the aortic segments sustained some injury, possibly a slight rupture, during the attack.

Dilatation and hypertrophy may, I am convinced, result from long continued palpitation. I have in several instances satisfied myself of the occurrence of temporary hypertrophy in the neurotic heart of young males; and dilatation and some hypertrophy are of frequent occurrence in the later stages of exophthalmic goitre.¹

Symptoms and physical signs.—The onset of neurotic palpitation is usually abrupt. In some cases, the exciting cause is something external to the organism (a loud noise, some cause of mental or emotional disturbance, etc.), which produces sudden stimulation of the sensory cerebral centres; in others the exciting cause is some internal irritation, such as

¹ In exophthalmic goitre the dilatation is probably due to other causes, such, for example, as degeneration of the muscular fibre, and not altogether to the excessive action of the organ.

dyspepsia or other visceral derangement. The attack may occur at any time. Some of the most severe cases which have come under my observation occurred during the night, the patient being awakened by the violent beating of the heart.

The most prominent symptom is the exaggerated pulsation of the heart; the organ may be felt thumping or knocking against the chest wall; in some cases the patient hears the beating as well as feels it. In severe cases, a feeling of intense anxiety or dread is at the same time experienced. Some præcordial distress is often present, and in exceptional cases, there is actual pain in the region of the heart. Violent throbbing pulsation in the peripheral vessels, noises in the ears, a feeling of tension and fulness in the head are common. The face is in some cases flushed; in others pale, expressive of anxiety, and perhaps bedewed with clammy sweat. A choking sensation in the throat (globus) or a feeling of syncope may be experienced; difficulty of breathing is sometimes present, but true dyspnœa is not usually observed. (In organic palpitation dyspnœa and other indications of embarrassed venous circulation are usually prominent symptoms.) When the cardiac action is at the same time intermittent or irregular, the patient is very sensible of the altered rhythm of the heart. The duration of the paroxysm is usually brief, but repeated attacks are apt to follow one another at short intervals. In some cases, the attack terminates as suddenly as it commenced. In a case, for instance, which I have recorded elsewhere the pulse suddenly dropped from 150 to 60, and the attack instantaneously disappeared. The termination may be attended with a copious discharge of pale, limpid (hysterical) urine. After severe attacks the patient is sometimes much exhausted; exceptionally fainting is observed.

During the paroxysm, the frequency of the cardiac contractions is, as a rule, much increased, the pulse may number 200 or even 250 in the minute; in some cases of organic palpitation the frequency of the heart's action is little if at all increased. On inspection, the area of cardiac impulse is seen to be increased; and violent beating is felt when the hand is placed over the præcordia. In exceptional cases, a thrill or

tremor can be perceived. The area of cardiac dulness is seldom altered to any appreciable extent. (This statement does not of course refer to organic palpitation in which the heart is so frequently dilated or hypertrophied). On auscultation, the first sound is unusually loud, ringing and metallic; the second sound is in some cases accentuated; but where the heart is acting very quickly it is usually less loud than in health or altogether effaced. Basic murmurs are sometimes observed; but occasionally a basic murmur, which was present when the heart was beating quietly, becomes effaced. (See the case reported by me in the *Lancet*, November 27, 1875, p. 764.) The heart's action and the pulse are, in most cases of neurotic palpitation, regular; but irregularities and intermission are not at all uncommon. In some cases, the pulse is full and bounding; in others, small and feeble. Fulness in the veins of the neck may be present.¹

Diagnosis.—In every case of palpitation the physician has to determine:—

1. Is the palpitation associated with organic cardiac disease, or is it neurotic in character?
2. If organic, what is the exact nature of the cardiac lesion?
3. If neurotic, what is the cause of the attack?

Step No. 1.—*Is the palpitation associated with organic cardiac disease, or is it neurotic?*

In order to determine this important point, attention must be directed to:—

1. *The physical condition of the heart.*—The size of the heart, the condition of the valvular apparatus, and the state of the cardiac muscle (as evidenced by the physical examination) must be carefully ascertained. The difficulties which attend the recognition of fatty degeneration and other structural lesions of the cardiac walls, when unattended with valvular disease or with enlargement, must be remembered. In most cases, even when the results of physical examination

¹ These statements have special reference to neurotic (functional) palpitation. In organic cases the character of the peripheral arterial and venous circulations depends of course upon the nature of the structural lesion.

are inconclusive, the other circumstances which I am about to mention usually enable us to come to a correct conclusion. Repeated careful examinations in different states of cardiac action are, however, in many cases necessary before a positive opinion can be formed. In fat women at the meno-pause, when fatty changes may be suspected; and in young males, in whom long continued palpitation sometimes, I believe, produces temporary hypertrophy and dilatation, the diagnosis may be especially difficult. In the latter case, provided that the valvular apparatus is healthy, that there is no disease of the kidneys, that there is no evidence of adherent pericardium, in short, no obvious mechanical cause of enlargement, we may, I think, reasonably conclude that the enlargement of the heart is the direct result of the palpitation, and that when the palpitation has subsided, it will, in all probability, disappear.

2. *The phenomena of the attack.*—In neurotic palpitation the subjective phenomena are out of all proportion to the mechanical derangement of the circulation, and there is no evidence of any structural lesion of the heart. In those cases in which the palpitation is associated with organic disease of the heart, symptoms due to mechanical derangement of the circulation are, as a rule, prominent.

3. *The effect of exertion on the heart.*—In the purely neurotic forms of palpitation, exertion produces little or no shortness of breath; whereas in the organic forms, exertion does produce shortness of breath and excites the palpitation. (In exophthalmic goitre and anæmia, shortness of breath and palpitation are induced by exertion.)

4. *The age, sex, and general condition of the patient.*—Both neurotic and organic palpitation may, of course, occur at any age; but neurotic palpitation is most common in young adults, whereas organic palpitation is most frequently observed after middle life. Neurotic palpitation is most common in females and in nervous males; organic palpitation occurs in both sexes, and in persons of all temperaments. Palpitation in an old person is always suggestive of organic disease. In women at the meno-pause, severe functional palpitation is

frequently observed. Middle-aged men who are depressed by business cares, or other causes of mental worry, often suffer from palpitation of the heart.

5. *The exciting cause of the attack.*—Neurotic palpitation is generally due to a start, fright, emotional disturbance or reflex impression arising in some of the peripheral organs; whereas organic palpitation is most frequently occasioned by muscular effort or other cause of increased arterial blood pressure. In anæmic cases, in which the nerves and muscular fibres of the heart are unduly irritable and the cardiac walls at the same time degenerated, an attack of palpitation may be induced in both ways, *i.e.* through the nervous system by emotional or other sudden stimulation, or mechanically by an increased strain being thrown on the heart.

Step No. 2.—If the palpitation is associated with organic disease of the heart, what is the exact nature of the cardiac lesion?

To this point, which must of course be determined by a careful and accurate investigation into all the facts of the case (the symptoms and physical signs), I need not further refer here. The physician must of course remember that the structural lesion of the heart may be either primary or secondary, and that the increased arterial blood pressure, which in so many cases is the direct exciting cause of the attack, is often due to extra-cardiac conditions. It is particularly important in all cases in which palpitation is associated with hypertrophy of the left ventricle, and in which the hypertrophy is not due to valvular disease, to examine the condition of the urine. Palpitation is in fact a very common symptom in the later stages of chronic Bright's disease, more especially of the cirrhotic form.

Step No. 3.—If there is no organic disease of the heart, what is the cause of the attack?

In some cases this question is easily answered, in others it can only be determined after a patient and careful investigation into the patient's habits and mode of life.

When the symptoms and signs of anæmia, more especially of cardiac anæmia (pallor of the mucous membranes, venous

hum in the neck, systolic pulmonary murmur, etc.) are present, a sufficient cause for the palpitation has been ascertained.

The condition of the thyroid and of the eyeballs should be carefully investigated, for palpitation is a most severe and troublesome symptom in exophthalmic goitre. It is important to remember, that the case may be one of exophthalmic goitre even although the eyeballs are not prominent, nor the thyroid enlarged. In typical cases of Grave's disease (exophthalmic goitre) all three symptoms (enlargement of the thyroid, prominence of the eyeballs, and excessive action of the heart) are present, but it is not very uncommon to find the prominence of the eyeballs wanting; probably in some cases the thyroid is not enlarged; and I have lately seen, in consultation, a gentleman whom a distinguished London physician thinks is suffering from Grave's disease, in whom the enlargement of the thyroid and the prominence of the eyeballs are both wanting, but in whom the cardiac symptoms are very prominent.

When the patient is not anæmic, and when the eyeballs are not prominent, and the thyroid not enlarged, the other possible causes of palpitation must be looked for.

In all cases, more especially in those in which the general health is below *par*, the apices of the lungs should be carefully examined, for palpitation is not uncommon in the earlier stages of phthisis.

In young women when there is no obvious cause, the probability that the palpitation is 'hysterical' must be kept in view, and other indications of hysteria inquired after. In young males the frequency with which palpitation is due to sexual excesses and irregularities, and to over-indulgence in tobacco, must be borne in mind.

When the palpitation cannot be ascribed to any of the above-mentioned causes, the fact that it is often due to over-indulgence in tea, to dyspepsia, to gout, and that it is sometimes caused by displacement of the uterus, ovarian irritation, worms in the intestine or other form of peripheral irritation must be remembered. The habits, dietary and mode of life of the patient must be carefully inquired into, and the condition

of the different organs which are likely to produce peripheral irritation investigated.

It must never be forgotten that mental anxiety, business worries, monetary cares, love disappointments, and the like, are very often at the root of the whole matter. The patient and his friends must therefore be discreetly questioned as to the existence of mental worry.

Prognosis.—The prognosis entirely depends upon the cause, and the facility with which that cause can be removed. Speaking generally, the prognosis is highly favourable in cases of pure neurotic palpitation. There are, however, some exceptions, exophthalmic goitre, for example, is an extremely intractable disease, and the palpitation due to that cause, though it can in many cases be relieved by appropriate treatment, is seldom completely cured. It is unnecessary, however, to go into details. I repeat, that the prognosis both in the organic and neurotic forms of palpitation must be entirely based upon the exact cause of the condition, and the possibility of removing that cause in each individual case. Palpitation is, in fact, one of the very best illustrations which can be given of the importance of a full and exact diagnosis. The prognosis and treatment of palpitation of the heart are mere guess work unless the exact cause of the condition has been ascertained.

Treatment.—The *first* object of treatment is to relieve the paroxysm. The *second*, to remove the cause and prevent the recurrence of the attack.

The relief of the paroxysm.—The external application of cold in the form of an ice bag to the præcordial region; the administration of stimulants (brandy, ammonia, ether) by the mouth; the inhalation of a few whiffs of chloroform; the subcutaneous injection of morphia; the administration of a full dose of bromide of potassium or of chloral; and the application of a galvanic or faradic current to the vagus in the neck, are the chief means which are likely to prove efficacious for the relief of the paroxysm itself. The attack is sometimes relieved by making the patient take a few deep

breaths, or by applying smelling salts, pepper, etc., to the nostrils, by anything in fact which produces reflex stimulation of the vagus. Valerian, assafœtida, and musk, are useful in some hysterical cases. When the palpitation is due to mental causes, the agitation must be soothed, calmed, and allayed, by judicious sympathy, counsel, or commands, in accordance with the individual peculiarities of the patient. In those cases (they are usually organic) in which the palpitation is due to a sudden increase of the arterial tension, the inhalation of nitrite of amyl is the most rapid and satisfactory means of obtaining relief; but this point will be more particularly referred to under the treatment of angina pectoris.

The prevention of the recurrence of the attack.—The first indication is, of course, to remove the cause. In organic cases this is for the most part impossible, but in many cases of neurotic palpitation it can be accomplished. The general health must be raised to the highest possible state of efficiency; all sources of mental anxiety must, if possible, be removed; the patient should lead a regular, and, as far as possible, outdoor life, keeping early hours, and avoiding excesses of all kinds; arsenic, and iron should be prescribed if there is any anæmia; a belladonna plaster over the præcordia often seems to be beneficial; bromide of potassium, digitalis, iron, arsenic, and strychnine are, in my experience, the most useful drugs. When the palpitation is clearly neurotic, the patient should be distinctly told that the heart is not diseased, and that there is no fear of sudden or immediate death. The application of a galvanic or faradic current to the vagus and sympathetic nerves in the neck is often distinctly beneficial. In hysterical cases, drug treatment is of minor importance compared to the general management of the case. The treatment of organic palpitation need not be specially considered, for it has been already described under the different organic cardiac lesions. (See mitral regurgitation, cardiac dilatation, etc.)

IRREGULARITY AND INTERMITTENT ACTION.

Irregular and intermittent action of the heart are often associated with palpitation, but frequently occur independently of that condition. Like palpitation, they are, in some cases, associated with organic disease ; in others purely functional and neurotic.

Organic irregularity.—In many cases in which the heart is diseased, but in which the lesion is stationary, and the symptoms slight or absent, the pulse intermits occasionally, and is irregular. Slight alterations of rhythm may be of little importance, and are very frequently observed in old people whose arteries are atheromatous and whose hearts are somewhat cirrhotic.

Irregular action of a more pronounced and serious description is very common in the advanced stages of mitral stenosis, and is also met with in the later stages of mitral regurgitation. It is of frequent occurrence in the advanced stages of all those affections in which the cardiac muscle is degenerated, and is observed therefore in many cases of pericarditis, myocarditis, fatty and fibroid degeneration ; it is very common in dilatation, and, in fact, in all those conditions in which compensation is failing or has given way. It is unnecessary to describe in detail the various forms of irregularity which are met with ; the most serious is that in which some of the ventricular contractions are so feeble that the blood-wave, which is propelled into the aorta, fails to reach the wrist.

Neurotic irregularity.—Many of the conditions which produce palpitation also produce neurotic irregularity of the heart, more especially over-indulgence in tea and tobacco, sexual excesses, and gout.

Symptoms and physical signs.—Organic irregularity is often unobserved by the patient. Neurotic irregularity and intermission are often very vividly experienced, the heart seeming

to 'stand still,' 'turn over,' etc., a feeling of palpitation being often at the same time experienced. In organic cases, the physical signs vary, of course, with the nature of the cardiac lesion. In purely neurotic cases, the intermittent or irregular action of the heart is the only evidence of cardiac derangement.

Diagnosis.—The steps in the diagnosis of intermittent or irregular action of the heart are the same as in the case of palpitation. We must *first* endeavour to determine whether the disordered action is due to or associated with structural organic disease of the heart. The condition of the valvular apparatus must be carefully investigated, and it is important to remember that in many cases of advanced stenosis of the mitral valve in which the cardiac action is extremely irregular, there is no presystolic murmur.¹ The presence or absence of cardiac dilatation must be specially noted, and the exact condition of the cardiac muscle, so far as is possible, ascertained. I must again emphasise the difficulty of diagnosing myocarditis, fibroid degeneration and fatty heart, and insist upon the necessity of repeated examinations before giving a positive opinion in cases of this description. After having made a minute physical examination of the heart, the effects of exercise should be noted, the presence or absence of other indications of cardiac derangement or disease ascertained, in short, all the other points to which I have referred in speaking of the diagnosis of palpitation investigated.

Prognosis.—The prognosis depends, as in the case of palpitation, upon the exact cause of the condition. Intermis-sion or simple nervous halt is, as a rule, of much less importance than irregularity. Each case must, however, be judged on its own merits, special attention being given to the condition of the heart and the exact cause of the cardiac derangement, and whether that cause is removable or not.

¹ The reader is referred to the description of mitral stenosis, its physical signs and diagnosis (see page 477), where these points are fully treated of.

Treatment.—The treatment of organic irregularity must be conducted in accordance with the nature of the cardiac lesion. In neurotic cases the same treatment which has been recommended for the treatment of neurotic palpitation is to be employed.

ANGINA PECTORIS.

Definition.—A neurotic affection characterised by paroxysms of intense pain in the region of the heart, and a terrible sensation of impending death. The pain usually radiates through the thorax, up to the left shoulder and down the left arm. The affection is in many cases associated with organic disease of the heart and the root of the aorta ; and in its typical and severe forms is apt to prove suddenly fatal.

Ætiology and Pathology.—The group of symptoms, included under the term angina pectoris, may in all probability be produced by a number of different causes. It is therefore difficult to give a satisfactory systematic account of the ætiology and pathology of the condition ; and it is, I believe, impossible to advance any single theory which will satisfactorily account for the phenomena of all cases. The essential feature of angina pectoris is pain in the region of the heart, in fact all observers are agreed in thinking that the cardiac pain must be referred to the area of distribution of the sensory cardiac nerves, *i.e.* to the heart itself.

Now all degrees of cardiac pain are met with,—but it is customary to limit the term angina pectoris to those cases—rarely met with before the age of forty—in which the pain is intense, and in which the terrible sensation of impending death is experienced ; while those cases of cardiac pain, which are of frequent occurrence in young persons, and in which the pain is usually less severe, are generally included under the term *pseudo-angina*. The former variety, which very frequently proves fatal, is very often associated with coarse pathological changes in the heart and root of the aorta, and very generally, I believe, with minute structural changes, usually degenerative in character, in the heart or blood-vessels. The latter seldom,

if ever, proves fatal, and is rarely associated with structural changes in the heart or vascular apparatus. The former may be appropriately termed the *organic* and the latter the *functional* form of angina pectoris. This division, into a serious and organic form and a comparatively trivial and inorganic form of the disease, is of practical clinical utility, and may be safely adopted, provided that it is clearly understood, that the two forms run one into the other, and that it is sometimes difficult or impossible to separate them at the bedside. It must also be remembered that the functional form of angina pectoris may, and I believe not unfrequently does, occur in later life, while the organic form is occasionally, though it must be confessed very rarely, met with before the age of forty. In order to understand the phenomena of angina pectoris, so far as our imperfect knowledge will at present allow, it will be well perhaps to consider cardiac pain as a whole, and to refer briefly to the construction, so to speak, of the sensory nerve apparatus of the heart.

In conditions of health, and when the heart is contracting quietly, we are unaware of the action of the organ. It is only, in fact, when the cardiac action is markedly deranged, either as the result of temporary and functional, or permanent and organic causes, that we are conscious of any cardiac sensations.

We become conscious of the cardiac action when the sensory nerve terminations in the heart and adjacent structures are more powerfully stimulated than they are in normal tranquil action of the organ ; under such circumstances an impression is conducted to, and registered by, the sensory perceptive centres in the cerebrum.

The exact course of the sensory nerve fibres, which convey impressions from the heart to the cerebrum, has not yet been definitely determined. In some of the lower animals, the frog, for example, sensory impressions seem to be conducted upwards by a special branch of the vagus, which has, therefore, been termed the sensitive nerve of the heart ; but it is probable that in man the channels of sensory conduction are by no means so limited ; the phenomena of angina pectoris seem, in fact, to prove that the cardiac branches of

the sympathetic contain sensory fibres. In man, sensory impressions are carried from the heart by the sympathetic fibres connected with the cardiac plexus, possibly also, as in the frog, by the cardiac branches of the vagus.

The sympathetic branches of the cardiac plexus pass, it will be remembered, to the first dorsal and three cervical ganglia and thence to the spinal cord. Sensory impressions, passing to the cerebrum from the heart through the sympathetic, pass *viâ* the spinal-cord, while sensory impressions passing from the heart through the vagus join the nerve centres at the medulla oblongata.

The exact course of the sympathetic fibres, which are supposed to conduct cardiac impressions upwards, in the spinal cord is unknown. The exact position, too, of the perceptive cerebral centre which receives such impressions is undetermined.

Now cardiac pain, *i.e.* pain referred to the heart itself may theoretically result from excessive stimulation of any part of the sensory nerve apparatus connected with the heart.¹ Theoretically, therefore, we may suppose that cardiac pain may be due to:—

(1) Irritation of the sensory nerve terminations in the wall of the heart itself, the sensory parts of the coronary plexus (sensory ganglia, if there are sensory ganglia, and sensory branches.)

(2) The sensory nerve fibres composing the cardiac plexus.

(3) The sensory conductors which connect the cardiac plexus with the sensory perceptive centres in the cerebrum. (In the case of the sympathetic conductors, the seat of irritation might theoretically be placed in (*a*) the branches connecting the cardiac plexus with the three cervical and first dorsal ganglia, (*b*) these ganglia themselves, (*c*) the branches connecting the ganglia with the spinal cord, (*d*) the spinal cord

¹ When a sensory nerve or sensory centre is irritated, the pain which results is referred in accordance with the law of '*eccentric projection*' to that part of the periphery from which the sensory conductors or sensory centres are in the habit of receiving impressions, rather than to the point of irritation itself.

itself, (e) the conductors above the spinal cord, *i.e.* between the spinal cord and the perceptive centre.)

(4) The perceptive cerebral centres themselves.

It is probable, I think, that cardiac pain is actually produced in most of these ways. But before proceeding to consider the exact manner in which the pain of angina pectoris is produced, let us turn for a moment to the records of *post-mortem* examinations, and see what lesions have been actually found after death. In a large proportion of typical cases of true angina pectoris (the form which I term organic, and which is apt to prove suddenly fatal), the coronary arteries have been found ossified. It is obvious that this condition is not of itself the cause of the angina, for ossification of the coronary arteries is an extremely common condition, and it is only in a small minority of the cases that symptoms of angina are observed.

Possibly in those cases in which angina pectoris does occur, the coronary nerves (which are so closely connected, in their course over the exterior of the heart with the branches of the coronary arteries) are implicated, just as the cardiac nerves which ramify over the arch of the aorta are affected in some cases of atheroma and chronic arteritis of the base of the aorta; but to this point I will again presently refer.

In many cases, the heart muscle is fatty or otherwise degenerated. In a considerable proportion of the cases of angina pectoris the base of the aorta is dilated or aneurismal; in some cases pericarditis has been observed; and in three cases which have been carefully examined by Lancereaux, Peter, and Bazy, a distinct change has been found in the branches of the cardiac plexus passing over the root of the aorta. Peter, for example, found in one of his cases slight dilatation of the base of the aorta, increased vascularity and thickening of the aortic coats, and evidence of old pericarditis at the base of the heart. The branches of the cardiac plexus, which were carefully dissected out from the fibrous adhesions surrounding the root of the aorta, were found on microscopical examination to be in a condition of chronic inflammation, the nerve tubes were separated by masses of connective tissue containing

numerous nuclei ; some of the nerve tubes were strangled, as it were, by this fibrous tissue, their myeline sheaths ruptured and transformed into an amorphous fatty mass.¹

In some cases, the cardiac valves (and more especially the aortic valves) have been diseased, in fact, all forms of cardiac lesion have been found. Very frequently the peripheral blood-vessels, as well as the aorta, are atheromatous. In some cases, the heart has been said to be healthy, but structural changes are, I believe, present, either in the heart or the arteries, or in both, in the vast majority, if not in all cases of angina pectoris which prove fatal.

Let us now endeavour to determine the exact manner in which the pain is produced in cases of angina pectoris. In most cases it is, I believe, due to irritation of the sensory nerve terminations in the wall of the heart itself. The exact manner in which the cardiac nerve terminations are irritated, has not yet been definitely determined. A very plausible theory is that which supposes that the irritation is due to spasmodic contraction of the cardiac muscle ; that the cardiac pain is in fact similar to the violent pain which is experienced in the calf muscles when they are spasmodically contracted as in ordinary cramp. Further, we know, as the result of actual experience, that cramp in the calf is most apt to arise when the muscle is exhausted and fatigued—all football players must be well aware of this fact. Further, Gaskell's observations have shown that when the vitality of the cardiac muscle is impaired by exhaustion, by injury, by malnutrition, the cardiac muscle loses its power of rapid contraction, and contracts with a prolonged tonic contraction in the same way as unstriated muscle. Again, clinical experience has shown that during the paroxysm of angina pectoris the systemic arterial tension is, in many cases, very notably increased.

Possibly, too, in those cases of angina pectoris in which the coronary arteries are atheromatous, the degenerative process which commenced in the inner coat of the coronary arteries (endarteritis deformans) has extended to the outer

¹ *Traité clinique et pratique des maladies du Cœur*, p. 673.

coat and implicated the coronary nerves. Under such circumstances the terminal nerve fibres in the cardiac walls would, we may theoretically suppose, be in an unusually irritable condition.

Now, taking all these facts in connection, we may, I think, with some probability theorise that in many cases of angina pectoris the sequence of events is as follows:—

Firstly, the blood pressure in systemic arterial circulation is suddenly increased, either as the result of changes arising in the central nervous system (vaso-motor centre), or in consequence of some external condition (sudden effort, exposure to cold, mental agitation, etc.), or reflex impulse arising within the body.

Secondly, in consequence of the sudden increase in the peripheral resistance, the left ventricle, or rather those fibres of the left ventricle, which are degenerated either as the result of imperfect blood supply (disease of the coronary arteries) or of degenerative changes in the cardiac muscle, are thrown into a temporary condition of spasm or cramp, which is attended with severe pain. It is of course quite possible, indeed probable, that in some cases in which the cardiac nerves are diseased, the attack arises independently of any sudden increase of the peripheral resistance. Should the supposition, which has previously been adduced as to the possibility of a lesion of the terminal branches of the coronary nerves, be correct,—powerful spasmodic contraction of the left ventricle not amounting to cardiac cramp, and which under ordinary circumstances would not be attended with cardiac pain, might possibly be sufficient to produce the condition.

Thirdly, this irritation of the terminal branches of the cardiac nerves, is reflected, *via* the sympathetic branches of the cardiac plexus and the spinal cord, to other parts of the periphery. Dr Allen Sturge, in a very suggestive paper, to which I am much indebted, and to which I would refer my readers who are interested in this subject, states, ‘that it is only when the commotion has begun in the cord, or passed up to the grey matter of the spinal cord from the sympathetic,

that any great extension (such as radiation to the arm or wall of the chest) can take place.'¹

We would naturally, of course, expect that the reflex impression would be first conducted to those parts of the periphery, which are more immediately connected with the spinal centres to which the cardiac branches of the sympathetic pass, and that when the irritation was very extreme, the impression might extend to other and more distant masses of spinal grey matter, and be reflected to other and more distant parts of the periphery. And such is in fact the case. In cases of true angina pectoris the pain radiates through the thorax, up to the left shoulder, and down the left arm, sometimes to the tips of the fingers; it not unfrequently passes up the left side of the neck, and in exceptionally severe cases it may pass down the right arm, or to the lower extremities, in fact to almost all parts of the body. The radiation to the left shoulder and left arm is so constantly observed in typical cases of angina pectoris, that it has been by some writers supposed that the primary lesion is situated in the spinal cord itself. Anstie, for example, thought that angina pectoris is probably due to 'a mainly unilateral morbid condition of the lower cervical and upper dorsal portion of the cord, liable of course to be seriously aggravated by such peripheral sources of irritation as would be furnished by diseases of the heart, and especially by diseases of the coronary arteries;' but with this opinion I cannot agree. In no case, so far as I am aware, has a lesion of the spinal cord been found, and it is extremely difficult to believe that any spinal lesion could be so constantly unilateral; the very fact, in short, which Anstie advanced against the radiation of the pain outwards from the heart, viz. the unilateral character of the brachial pain, seems to me strongly opposed to his view. The unilateral character is, I believe, due to the fact that the irritation of the cardiac nerves is in most cases limited to the nerves of the left ventricle, for it is this cavity which has to overcome the sudden increase in the peripheral arterial resistance which is often the starting point of the attack. When

¹ *Brain*, January 1883, p. 496.

the pain, which has originated in the region of the heart, and has radiated to the left arm in the usual way, passes, as it sometimes does (but only in severe or exceptional cases), to the right arm, the peripheral irritation has been sufficiently severe to pass over to the opposite side of the spinal cord. In other cases the radiation of the pain to the right arm is, I think, to be explained either by supposing that some of the fibres of the right ventricle have also become affected, and the nerve terminations in the walls of that cavity have become irritated ; or, that the primary seat of the lesion (*i.e.* of the irritation) is outside the heart in the coronary plexus,—a lesion, for example, of the root of the aorta.

It has also been supposed by some writers that the paroxysm of angina pectoris can be produced by a sudden diminution of the blood supply to the heart itself, the coronary arteries sharing of course in the general vascular spasm which is the cause of the increased arterial tension to which I have previously referred as the exciting cause of the attack.

In other cases of angina pectoris the primary lesion is probably extra-cardiac. I have previously stated that in many cases the base of the aorta is diseased, and that in some cases of this description an actual lesion of the branches of the cardiac plexus, which ramify over the arch of the aorta, has been demonstrated. The same explanation, *i.e.* direct irritation of the branches of the cardiac plexus, would of course satisfactorily account for the occurrence of angina-like pain in cases of pericarditis. (It must, of course, be remembered that in those cases in which the branches of the cardiac plexus are involved in a chronic lesion, the degenerative changes would probably extend downwards to the peripheral terminations of the cardiac nerves in the heart, and that the partially degenerated nerves might be more irritable than in health. Under such circumstances, cardiac spasm, produced in the manner I have previously endeavoured to explain, would be very likely to cause an attack of angina pectoris.) Possibly too, in those cases in which a lesion of the cardiac nerves surrounding the aorta cannot be demonstrated, there may be lesion of the branches of the coronary

plexus, or degenerative changes in the coronary arteries implicating the coronary nerves which ramify so extensively over these vessels. Such a supposition would go far to explain the frequent association of disease of the coronary arteries with angina pectoris.

In other cases of angina pectoris, the primary lesion is possibly situated in the nerve-centres. This is probably, I think, the cause of the angina-like attacks which are sometimes met with in hysterical women. Under this head also I would place many of the cases of so called pseudo-angina.

Cases of functional angina pectoris, or pseudo-angina, in which there is no structural lesion of the heart or other parts of the vascular apparatus, might very appropriately be termed cases of cardiac neuralgia. Cases of pseudo-angina are of frequent occurrence in anæmic and hysterical women, and in young males who are exhausted by sexual or other excesses, or who over indulge in tobacco.

The organic form of angina pectoris is often met with in gouty subjects, and is popularly included with other conditions under the term 'gout in the stomach.' Gout, it will be remembered, is one of the conditions which produce atheroma, and therefore disease of the aorta and coronary arteries. In short, cardiac neuralgia is produced by the same causes which produce neuralgia in other parts of the body. Why the neuralgia affects the nerves of the heart it is usually impossible to surmise, just as it is usually impossible to ascertain what are the causes which determine the locality of the lesion in neuralgia of the fifth nerve for example.

Symptoms and Physical Signs.—Angina pectoris, of which it will be only necessary to describe the more severe forms, is a paroxysmal affection, and is seldom observed before the age of forty. The patient is usually a male, for the atheromatous condition of the aorta and coronary arteries, which is such an important factor in the production of the organic form of the disease, is comparatively seldom observed until after middle life, and is much more common in men than in women. The exciting cause of the paroxysm is in many

cases bodily exertion (walking too quickly, climbing a hill, etc.), mental excitement, straining at stool, exposure to cold, or other causes of increased arterial blood pressure. In some cases no definite exciting cause can be ascertained. The attack commences with pain in the region of the heart; in well marked cases the pain is intense, it radiates through the thorax to the spine, and usually extends up to the left shoulder and down the inner side of the left arm, often to the tips of the fingers. It also radiates up the left side of the neck; less frequently it extends to the right shoulder and down the right arm; and in exceptional cases it has been known to pass to the lower extremities, and seemed in fact to shoot all over the body. In rare cases the pain is more marked in the right arm than in the left; in cases of this description, and indeed in most cases in which the extension is to the right as well as to the left, it is probable, I think, that the lesion will be found at the root of the aorta, or in the aortic arch, rather than in the heart itself. It is obvious, on anatomical grounds, that a lesion of the aorta implicating the branches of the cardiac plexus, will in most cases involve the branches of the sympathetic proceeding from the right side of the spinal cord as well as those passing to the left. In addition to this intense pain, a terrible feeling of impending death is experienced; in many cases the chest feels as if it were fixed in a vice, the patient dreads making any movement, and does not dare to take a deep breath; the countenance is expressive of the terrible agony and dreadful sensation of impending dissolution which the patient is experiencing, the features are generally at the same time pinched, the colour pale, and the face in some cases covered with a cold clammy perspiration; in short, in most cases the features are at the same time expressive of suffering, fear, and collapse.

The condition of the pulse varies in different cases; usually it is small, quick, and hard, and sphygmographic observations have shown that in many cases the arterial blood pressure is very markedly increased. In some cases the pulse is irregular. In a case of hysterical angina pectoris which was under my care for some time, the patient lay for some days in a state

of collapse, in which the skin was cool and pale, and the pulse beating at the rate of 180–200 per minute; severe and very frequently recurring pain, which presented all the usual characteristics of angina-like pain, was experienced in the præcordial region; the pulse tension was not increased, and these attacks were not relieved by the nitrite of amyl. In some cases the action of the heart has been said to be natural and the pulse tranquil. In others the pulse has been found to be slower than normal during the attack.

The respirations are usually, but not invariably increased in frequency; as a rule the respiratory movements are more superficial and more shallow than in health, but in some cases dyspnœa is observed. Vomiting has been noted in some cases, in others vertigo, and occasionally more serious indications of derangement of the cerebral nerve centres, such as spasmodic twitchings, or even general epileptiform convulsions, occur.

The paroxysm is usually of short duration (a few minutes to a quarter of an hour) but in exceptional cases it lasts for a longer time. As the attack passes off, wind is frequently eructated from the stomach, and in some cases there is a copious discharge of pale, limpid urine.

After a severe attack, the patient is naturally much exhausted, he looks terribly shaken; for days, or even longer, his whole expression and bearing may be indicative of the dreadful nature of the ordeal which he has just passed through. After the pain subsides, a feeling of numbness (anæsthesia), and sometimes of loss of motor power (paresis) is experienced in the affected arm, *i.e.* usually the left arm. Occasionally, too, there is numbness and loss of sensibility in the skin of the præcordia. Sometimes hyperæsthesia, and not anæsthesia, is observed.

Should the physician have an opportunity of examining the heart during the attack, the action will, as a rule, be found to be quickened, and the impulse and sounds weaker and more distant than in health. In some cases the action of the heart has been said to be quite normal. In those cases in which valvular lesions, pericarditis, and other organic changes,

attended with well-marked physical signs are present, the usual indications of those lesions will of course be observed.

After a sufficient time has elapsed to allow of recovery from the exhaustion, collapse, and after-effects of the attack, —and some days or even weeks may be required for complete recovery—the patient is restored to his previous condition of health. It is very essential after convalescence has been fairly established, to make a minute and careful examination of the heart and blood-vessels. Special attention should be given to the condition of the heart itself, the base of the aorta, and the peripheral arteries. Evidence of dilatation of the root of the aorta, and of general atheroma—conditions which suggest the presence of atheroma of the coronary arteries—will, in many cases, be detected. In others, the usual symptoms and signs of aortic regurgitation will be observed; and in a few cases the physical indications of fatty heart. In many cases, the heart itself appears to be healthy, but the peripheral blood-vessels and the condition of the radial pulse are suggestive of commencing arterial degeneration; the *arcus senilis* is not infrequent in such cases, and though *per se* of little value as a diagnostic of any special cardiac lesion, it is suggestive of degenerative changes in the vascular system. In some cases, more especially in gouty subjects, the condition of the urine may suggest cirrhotic changes in the kidney.

In pseudo-angina, or functional angina as I prefer to term it, there are no indications of cardiac, aortic, or vascular disease.¹

The frequency with which the paroxysms recur varies considerably in different cases. Quite exceptionally there is no return of the attack; very frequently, several months, it may even be years, separate the first and second attacks; the interval between the second and third attacks is usually shorter; and in most cases as the disease goes on, the

¹ I refer of course to typical cases of pseudo-angina, such as are met with in young males and hysterical females, and do not include the præcordial pains (not amounting to true angina pectoris) which are met with in some cardiac affections —aortic regurgitation for example.

paroxysms become more and more frequent, and are more and more easily excited. Ultimately the slightest exertion, such as stooping to put on boots, may produce a paroxysm. The severity of the attacks too is apt to increase as the case progresses.

Diagnosis.—The diagnosis of severe cases of angina pectoris does not present any difficulty, the character of the pain, the terrible nature of the suffering, more especially the dread feeling of impending death, are quite distinctive. It is very important, however, to remember, *firstly*, that in grave cases of angina pectoris the pain is not always typical and severe; and *secondly*, that severe pain in the region of the heart, apparently identical with the pain of the organic form of the disease, may be due to purely functional causes. When the attack is not a very severe one, and more especially when the physician has not had an opportunity of witnessing a paroxysm, it may be extremely difficult to form an opinion as to the true nature of the case.

Angina-like pain should, however, always excite grave apprehension, and in all cases, in which such pain is experienced, the physician must endeavour to determine whether he has to do with the organic or the functional form of the disease. In attempting to decide this very important question, attention should be directed to the following points:—

1. *The condition of the heart and vascular apparatus generally.*
2. *The age and sex of the patient.*
3. *The severity and character of the symptoms.*

Organic disease of the heart, more especially aortic valvular disease; simple or aneurismal dilatation of the aorta; atheroma of the superficial vessels; the presence of an *arcus senilis*, and in fact any other indications of degenerative changes, more especially of degenerative changes likely to affect the heart and blood-vessels; the facts that the patient is over forty years of age, and is a male, that the pain was severe, that it radiated down the left arm, and that the attack was accompanied by the feeling of impending death, are all strongly suggestive of the organic and serious form of the disease.

When on the contrary the physician is satisfied that there is no disease of the heart or blood-vessels, when the patient is a female, and especially when in addition the patient (whether a male or a female) is under forty years of age, and when there is no reason to suspect degenerative arterial changes, the diagnosis of functional angina pectoris may be ventured upon.

I must, however, caution the observer against too hastily deciding in favour of the functional form of the disease if there is any reason to suspect, either from the age of the patient, or any other fact, that degenerative arterial changes may be present. It must never be forgotten that the coronary arteries may be atheromatous in cases in which there are no physical signs of cardiac disease, no evidence of disease of the aorta, and even in which there is no distinctive evidence of atheroma of the superficial vessels. It must also be remembered that it is often impossible to detect a small aneurism at the root of the aorta ; while, as we have previously seen, the diagnosis of fatty heart—a condition which may be attended with angina pectoris—is often most difficult.

Prognosis.—The prognosis of the organic form of angina pectoris is most grave, and it must never be forgotten that the patient may die during the attack. The opinion must to a large extent be based upon the condition of the heart and arteries, the frequency and severity of the paroxysms, the circumstances and surroundings of the patient, his capabilities of following out the treatment which is recommended, more especially his capabilities of avoiding all causes of sudden increased arterial strain, his mental temperament, etc. It must always be remembered that a patient who has once had a severe and typical attack of angina pectoris may at any time have a second, which may prove immediately fatal. The tendency is, in fact, to the more frequent recurrence of the attacks as the case progresses, and for the severity of the attacks to increase rather than to diminish ; on the other hand, cases are sometimes met with in which there is no recurrence. Provided then that the cardiac or vascular disease

is not serious and does not progress, and that the patient is able to avoid the exciting causes of the attack, he may live for years, and enjoy a fairly comfortable, and even active existence. I have met with two or three cases, in which several attacks of typical and severe angina pectoris (which there is every reason to believe were associated with degenerative changes in the aorta and probably in the coronary arteries), have occurred, and in which the patients have not only continued to live for some years, but have also continued to lead active, and indeed laborious professional lives. Such cases are, however, the exceptions not the rule. In all cases of typical angina pectoris after forty years of age, the prognosis should be most guarded, more especially if there is any evidence of degenerative arterial change.

The prognosis of the functional form of angina pectoris is favourable.

Treatment.—In treating any paroxysmal affection, we must endeavour:—*Firstly*, to relieve the paroxysm, and *secondly*, to prevent its recurrence, and to cure the condition on which it depends.

The relief of the paroxysm.—To Dr Lauder Brunton belongs the great merit of having discovered that the arterial blood pressure is increased in many cases of angina pectoris, and that by the administration of nitrite of amyl inhalations, the paroxysm may often be steadily and completely relieved. Subsequent observations have abundantly confirmed Dr Brunton's discovery, which is one of the most important therapeutic advances of our time. The drug acts by suddenly reducing the arterial tension or spasm on which the paroxysm primarily depends. Since angina pectoris may be produced by several different conditions, nitrite of amyl is not, of course, a specific. It may be expected to be effective in those cases in which the blood pressure is high during the paroxysm, and in which the main object of treatment is to produce a rapid reduction of the increased pressure. Three drops should be poured upon a handkerchief or piece of blotting paper and inhaled during the attack. In many

cases a larger dose (5-10 drops) is required, but the smaller quantity should first be tried, for some people are unusually susceptible to the drug. A patient who has once had an attack of angina pectoris should always carry some nitrite of amyl about with him; the remedy may be had in glass capsules, and should a paroxysm come on, it is only necessary to break one of the capsules in the handkerchief and inhale the drug.

Another most valuable remedy, which also acts by reducing the arterial tension, is nitro-glycerine. We are indebted for most of our knowledge with regard to the value of this drug in angina pectoris to Dr Murrell. One drop of a 1 per cent. spirituous solution, or a lozenge containing the $\frac{1}{100}$ th part of a grain may be given three times daily. The remedy is particularly useful in those cases in which the arterial tension is habitually high, and in which repeated paroxysms of angina are apt to occur. It is more valuable, therefore, as a preventative of the paroxysms than for the relief of the paroxysm itself; it acts, however, in both ways. It is inferior to nitrite of amyl for the rapid reduction of arterial blood pressure; for the relief of the paroxysm itself, therefore, nitrite of amyl is to be preferred.

In those cases in which nitrite of amyl inhalations fail to relieve the attack, and in cases in which it is not at hand, recourse must be had to other measures. The administration of a full dose of ether, brandy, ammonia, or other diffusible stimulant; the subcutaneous injection of a full dose of morphia; and more especially the inhalation of chloroform or ether, are the measures which are most efficacious. The application of a mustard blister to the præcordial region is often useful. Professor Peter speaks highly of local depletion by means of leeches applied to the præcordia.

The prevention of the recurrence of the attack.—In order to prevent the recurrence of the paroxysm, it is essential to avoid the exciting causes of the attack, to raise the general health to the highest possible state of efficiency, and to treat any organic lesion of the heart, blood-vessels, or other organs which may be present.

When the arterial tension is habitually high, nitro-glycerine should be given for several days in the manner recommended above ; and the general measures, such as occasional purgation, restriction of the food and drink, etc., which are useful in the treatment of chronic conditions of high arterial tension, employed.

Patients who have had an attack of angina pectoris should be impressed with the gravity of the affection (though in severe cases any such advice is quite unnecessary, for the agony of the attack is so fearful that they know full well of themselves that a recurrence may prove fatal), and of the necessity of avoiding anything which is likely to suddenly increase the blood pressure. All sudden efforts must be carefully avoided. Persons of an excitable, irritable, and hasty temper are very difficult to treat, and it is all the more important to impress them with the absolute necessity of avoiding all causes of mental excitement, and of living, so far as their disposition will permit, placid, quiet, and cheerful lives. The diet should be easily digestible, but at the same time nutritious, the quantity of food taken at a meal should be strictly moderate ; in short, great care must be taken to avoid distention of the stomach, flatulence, dyspepsia, etc. Nervine tonics and anti-neuralgic remedies are in many cases most useful. Arsenic is a valuable remedy both in the organic and functional forms of the disease. Quinine and iodide of potassium may, in some cases, be given with advantage.

Duchenne claimed to have cured angina pectoris by the local application of the faradic current ; and Eulenberg speaks highly of the advantages of galvanism. 'If correctly employed it is probably,' he states, 'a remedy of chief importance, and perhaps the only direct remedy for angina pectoris. But the nature of the symptoms will direct us when to select methods of application which produce reflex excitation of the regulator nerves of the heart, and when to prefer direct galvanization of the cervical sympathetic and cervical vagus. I have only been able to use the former procedure in three cases of accelerated action of the heart without organic disease,—but could not continue the application long

in either instance. The effect was distinctly good; the attacks became less severe; and in one case, ceased entirely, while previously they had made their appearance almost daily. In a fourth case, recently brought under treatment, the attacks have become rarer and milder. The method used consists in the application of strong stable currents, rising to the number of 30 elements; the positive pole, with a broad surface, is placed upon the sternum, while the negative is placed on the lower cervical vertebræ. Von Heubner has lately obtained a permanent cure by similar methods in a case which seemed to have a rheumatic origin. He placed the positive electrode upon the fossa supra sternalis, and the negative upon the cervical ganglia of the sympathetic, on both sides in succession; then he placed the positive pole upon the lowest cervical ganglion, and the negative upon the sensitive spots, at the angles of both shoulder-blades. At first only very weak currents, of from 4 to 6 elements, were borne. The attacks ceased from the first session, and did not return; and by degrees it became possible to use stronger currents, of from 8 to 10 elements.¹

Electric treatment is most likely to prove useful in the functional, and more purely neurotic forms of the disease. In the organic forms, one of the most important points is to treat the cardiac or arterial lesion which happens to be present. The greatest attention must always be given to this point, but it is unnecessary to enter into details which will be found under the heads of aortic valvular lesions, aneurism, atheroma, pericarditis, fatty heart, etc.

In treating the functional forms of angina pectoris, the condition of the general health must be carefully attended to. Careful inquiry must be made into the habits of the patient, excesses of all kinds forbidden, anæmia treated, in short, the general and special treatment which is advisable in any case of ordinary neuralgia carried out.

¹ *Ziemssen's Cyclopædia*, vol. xiv. p. 54.

CHAPTER VIII.

DISEASES OF THE THORACIC AORTA ACUTE AORTITIS. ATHEROMA.
GENERAL DILATATION. ANEURISM. COARCTATION OF THE AORTIC
ARCH.

THE more important affections of the thoracic aorta which call for consideration are :—

1. Acute inflammation, or acute aortitis, as it is termed.
2. The chronic inflammatory and degenerative processes and their results, amongst which the most important are atheroma, general dilatation, and aneurism.

The reader is particularly recommended, before commencing the study of the individual diseases of the aorta, to refer to the description, which has been previously given, of the anatomical relations of the aorta and of the clinical methods by which its condition is investigated (see page 224).

ACUTE AORTITIS.

Acute inflammation of the outer coat of the aorta is sometimes observed as the result of inflammation of the surrounding parts—the pericardium and mediastinal tissues. Acute inflammation limited to the aorta, and involving the whole thickness of its wall, is of very rare occurrence ; no case, either during life or after death, has come under my own observation, which I could distinctly recognise as answering to the descriptions which have been given of the condition.

Symptoms and physical signs.—Fever, with rigors and general uneasiness, throbbing pulsation, and (in some cases) severe pain in the region of the aorta, tumultuous action of the heart, disturbances of breathing, and symptoms due to embolic infarctions in distant parts, a tendency to syncope, and apprehension of impending death, are some of the more

prominent symptoms which have been noticed in cases of supposed acute aortitis.

The *physical signs* are indefinite, and are often complicated with those of cardiac disease; throbbing pulsation of the aorta, irregular and tumultuous action of the heart, a systolic aortic murmur, and the secondary physical signs due to embolic infarctions, are probably the most important.

Diagnosis.—Acute aortitis can seldom, if ever, be positively recognised during life. According to Walshe, ‘pain, thrill, and pulsation in the course of the vessel, with arterial murmur coasting the spine, and answering in localisation neither to a murmur of the aortic nor of the mitral valves, would be the conditions, coupled with great distress and pyrexia, most nearly warranting the diagnosis of the disease. But,’ he adds, ‘it is needless to point out the varieties of states that might simulate the entire series, except the aortic murmur, and in respect to this murmur the possibility of its depending upon chronic disease proclaims the necessity of caution.’¹

Treatment.—The application of leeches, and cold in the form of icebags, over the course of the aorta, together with the internal administration of antipyretic remedies, and of opium, aconite, digitalis and belladonna, in accordance with the special requirements of each case, seem the measures most worthy of recommendation. The same treatment, which has been recommended for acute endocarditis, may also be employed.

ATHEROMA AND GENERAL DILATATION OF THE AORTA.

Atheroma of the aorta is of very common occurrence, and is, in many cases, associated with disease of the aortic valves, general or local dilatation (aneurism) of the aorta, and atheroma of the coronary arteries and peripheral vessels.

Ætiology and Pathology.—General atheroma, except the form which results from syphilis, is essentially a senile change,

¹ *Diseases of the Heart*, fourth edition, p. 472.

and is seldom seen in an advanced degree before the age of forty-five, though fatty spots at the base of the aorta are sometimes met with in quite young subjects, and general dilatation and inflammation of the aortic arch may develop in comparatively young persons, as the result of strain and aortic incompetence.

Males are much more frequently affected with atheroma than females.

The great causes of the condition are strain (more especially frequently repeated and sudden increases of the blood pressure), syphilis, alcoholic excesses, gout, rheumatism, exposure to cold, and depressing influences of all kinds. The condition seems sometimes to be hereditary.



FIG. 264.—Section through a gelatinous spot in a case of atheroma. ($\times 180$.)

a, free surface of the aorta at the summit of the gelatinous patch ; *b*, free surface of aorta at the side of the gelatinous patch ; *c*, elastic lamina ; *d*, round cells ; *e*, spindle cells ; *f*, collection of oil globules in the midst of fibrous tissue.

Pathology.—In the earlier stages of the process, opaque, yellow patches, or raised gelatinous-looking nodules of a pinkish-grey colour, are seen scattered here and there, on the interior of the aorta. On *microscopical examination*, the gelatinous patches are found to consist of spindle-shaped cells, and of round cellular elements which stain deeply with picro-carmin, and which are for the most part arranged in rows, parallel to the long diameter of the vessel. (See fig. 264.)

As the disease advances, collections of fatty particles are seen, more especially in the deeper layers of the inner coat; ultimately the tissue is in places completely destroyed, and the broken down fatty debris is collected in cavities, to which the term atheromatous foci has been given; calcareous particles are at the same time deposited (see fig. 265); the middle and outer coats are generally infiltrated here and there, with round cells, and the nutrient vessels of the aorta are often found to be undergoing obliteration, *i.e.* to be affected with endarteritis obliterans.

The naked-eye appearances which the aorta presents are now striking; the vessel is usually more or less dilated, the dilatation being sometimes general and uniform, in other cases partial and local; its interior is studded here and there with hard projecting nodules, some of which have the consistency of cartilage, others being hard and dense like bone, the latter, which are in reality calcareous plates, are, for the most part, thin and brittle; fissures, cracks, and small irregular ulcers, which communicate with atheromatous foci in the subjacent tissue, are frequently seen in the interior of the aorta; not unfrequently the sharp edge of a thin calcareous plate projects through the ruptured lining membrane, and the blood is seen to have made its way into the atheromatous cavities and between the coats of the artery, forming a small dissecting aneurism.

Pathological physiology.—The effect of the atheromatous process is to impair the elasticity and resisting power of the aortic wall; local or general dilatation of the vessel is consequently very apt to occur under the influence of the

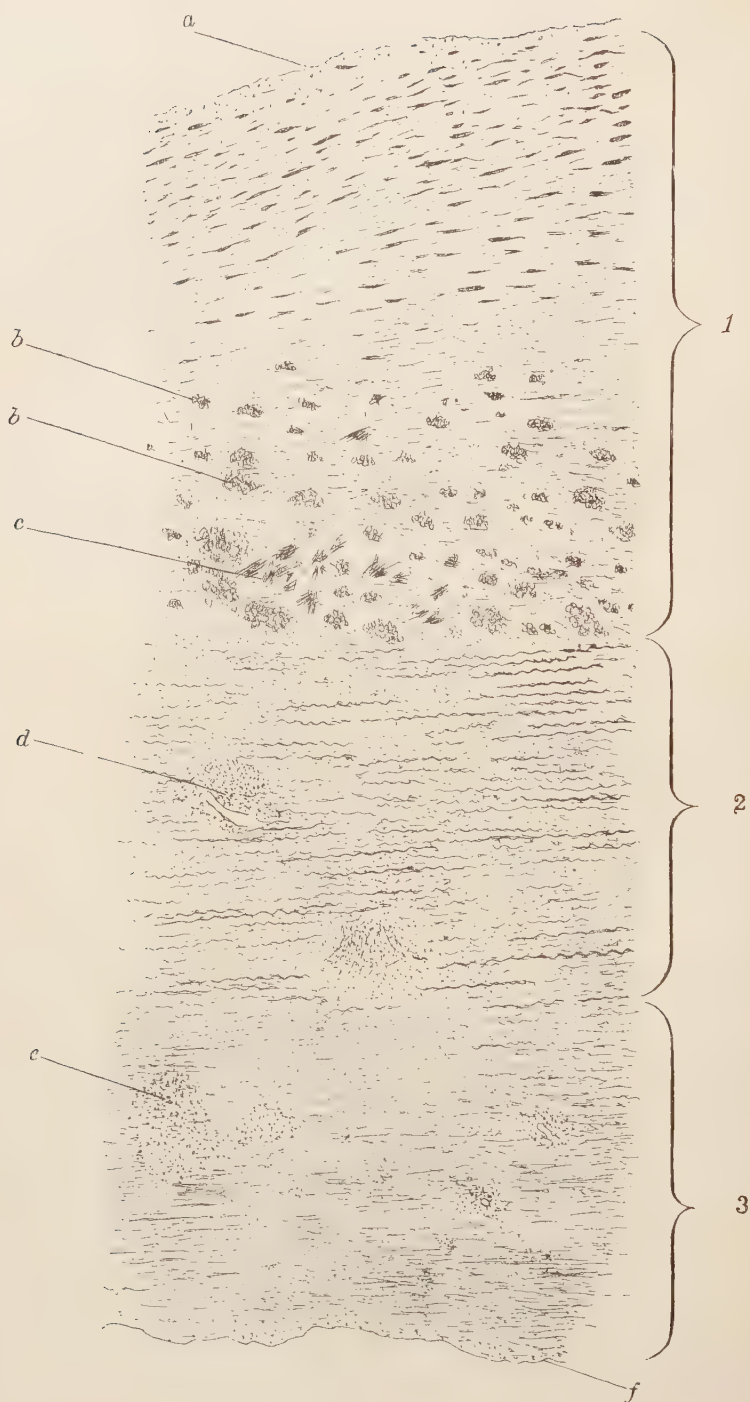


FIG. 265.—Section through the whole thickness of the aorta in atheroma.
(\times about 40.)

1, inner; 2, middle; and 3, outer coats; *a*, free surface of lining membrane; *b*, *b'*, fatty particles; *c*, fat crystals; *d*, leucocytes surrounding vessel of middle coat; *e*, leucocytes in outer coat; *f*, outer surface of vessel.

blood-pressure. But the effects of the atheromatous process are seldom limited to the aorta itself ; in many cases the aortic valves are involved, and the peripheral blood-vessels and coronary arteries similarly affected ; secondary changes (hypertrophy, dilatation, and in some cases mitral incompetence) are often, therefore, present in the heart.¹

Symptoms.—Symptoms may be entirely wanting, and this is more particularly the case when the disease is confined to the aorta itself, *i.e.* when the peripheral vessels are not affected, when the aortic valve is not incompetent, and when the coronary circulation is not interfered with.

When the peripheral blood-vessels are affected, symptoms and signs of defective cerebral circulation (giddiness, fainting fits, irritability of temper, defective cerebration, etc.) are of frequent occurrence.

When the aortic arch is dilated, shortness of breath and other symptoms of intra-thoracic pressure may be present.

When the root of the aorta is affected, and more especially when the coronary arteries are involved, or the branches of the coronary plexus which ramify over the root of the aorta implicated, pain of an angina-like character or attacks of true angina pectoris may occur.

Embolic symptoms are in some cases observed, portions of fibrine, which have been deposited on the roughened surface of the aorta, being detached and carried to some distant vessel.

When the aortic valve is incompetent, when the left ventricle has become dilated, or the mitral valve incompetent, other symptoms will of course be observed.²

Physical signs.—In those cases in which the aortic arch is of normal size, and in which there are as yet no secondary

¹ The secondary cardiac changes met with in atheroma of the aortic arch, may be due to:—(a) the difficulty which the blood has in passing through the rigid vessels ; (b) aortic incompetence, which is often present ; (c) obstruction to the coronary circulation, which may be present in consequence of narrowing of the orifices or atheromatous disease of the trunks of the coronary arteries.

² See the articles on aortic regurgitation, dilatation of the left ventricle, etc.

alterations in the left heart, physical signs may be entirely wanting; in cases of this description the aortic second sound is very often accentuated, in consequence of the obstruction to the blood-flow through the aorta and peripheral vessels.

When the aortic arch is dilated, increased dulness over the manubrium sterni and pulsation in the supra-sternal notch can usually be detected; a systolic murmur is in many cases audible, and a systolic thrill can sometimes be felt, over the course of the aorta; in these cases, the aortic second sound is often loudly accentuated.

When the aortic valve is incompetent, and when secondary changes (hypertrophy, dilatation, etc.) have taken place in the heart, other physical signs will of course be present.

In those cases in which the atheromatous process involves the smaller arteries, the radials, temporals, and other superficial vessels may stand out like tortuous cords; the thickening and rigidity of their coats can be felt when they are compressed by the finger; the tidal wave in the sphygmographic tracing is unusually prominent. (See fig. 266.)

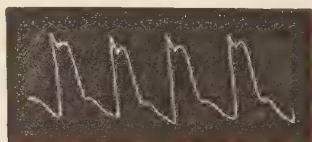


FIG. 266.—*Atheroma and Aneurism of Aortic Arch.*—J. D., æt. 52, admitted to Newcastle Infirmary 21st February 1878, suffering from aneurism of the ascending portion of the aortic arch and atheroma. The tidal wave is very strongly marked. There was no perceptible difference between the two pulses.

Diagnosis.—The opinion must be entirely based upon the physical condition of the aorta and of the peripheral vessels. The facts, that the patient has passed the prime of life; that indications of tissue degeneration, such as the *arcus senilis*, are present; a history of syphilis, gout, alcoholic excess, and exposure to want and privation, afford corroborative evidence in doubtful cases.

Prognosis.—So far as is at present known, atheroma is an incurable condition ; the opinion as to the probable duration of the case must be chiefly based upon: (1) the extent of the disease, as evidenced by the condition of the aorta and superficial vessels ; (2) the circumstances, habits, and surroundings of the patient—whether he is able to lead a quiet ‘strainless’ life, his alcoholic tendencies, etc. ; (3.) the condition of the aortic valves and of the heart.

The opinion must always be guarded, for sudden accidents which it may be impossible to foresee (such as the bursting of a cerebral blood vessel, embolic plugging of a cerebral vessel, the rupture of an aortic aneurism too small for detection, or the occurrence of cardiac syncope), may at any time occur.

Treatment.—The main objects of treatment are to keep the circulation as quiet as possible, and to maintain the condition of the general health. Everything likely to increase the blood-pressure within the aorta must be carefully guarded against, but the rigid system of rest, which will afterwards be recommended for the treatment of aortic aneurisms, is neither necessary nor desirable. The treatment must, of course, be conducted in accordance with the condition of the heart, and the special requirements of each individual case ; when, for instance, symptoms and signs of cardiac dilatation and failure arise, digitalis and stimulants, remedies which are to be avoided in the earlier stages of the case, must be prescribed. It is unnecessary to enter into details which the reader who is familiar with the treatment of cardiac valvular lesions and of aortic aneurisms, will readily supply for himself.

ANEURISM OF THE THORACIC AORTA.

Aneurism of the thoracic aorta is a common condition, and is of great practical and clinical interest.

Varieties.—Aneurisms of the thoracic aorta, which have not perforated the chest, are almost invariably *true* aneurisms, that is to say, the aneurismal sac is composed of one or more of the three natural coats of the aorta (the interna, the

media, and the adventitia). After the sac perforates the chest wall, blood escapes beneath the muscles and subcutaneous tissues, and a *false* aneurism is formed, external to the cavity of the thorax, in which the *true* aneurism is situated. Occasionally a false aneurism is formed within the cavity of the chest or abdomen, by the rupture of the true sac and the escape of blood into the surrounding parts. It is rare to meet with a false aneurism of this description within the chest, and it is only likely to occur in the presence of pleuritic adhesions, which place a limit on the amount of blood which can be extravasated. Rupture into a healthy pleura speedily proves fatal.¹ In some cases, the dilatation of the aorta is general, and the aneurism assumes a *globular* or *fusiform* shape; in others, and these are the most frequent, the bulging is partial, and the aneurism is said to be *saccular*. (See fig. 267.)² Occasionally the aneurism bursts into a vein, to these cases the term *arterio-venous* aneurism is given. When the internal coat is ruptured, and blood escapes between it and the media, or between the different layers of which the media is composed, the aneurism is called *dissecting*. A combination of these different forms is not unfrequently met with (see figs. 268 and 269), in which there are three aneurisms, one a good example of the globular or fusiform variety, and the other two sacculated).

Ætiology and Pathology.—The great causes of aneurism of the thoracic aorta, indeed of all internal aneurisms, are, *firstly*, local weakness of the arterial wall, and *secondly*, increased arterial blood pressure.

The most common cause of local weakness of the aortic wall is chronic endarteritis (atheroma), but any degeneration or inflammation either of the internal, the middle, or the outer coat, will produce local weakness, and will therefore predispose to the formation of aneurism. Degeneration of the

¹ Occasionally the patient survives for a few days.

² Aneurisms of the abdominal aorta are not described in this work, but this specimen is represented because it is more typically *saccular* than any aneurism of the thoracic aorta which I possess.

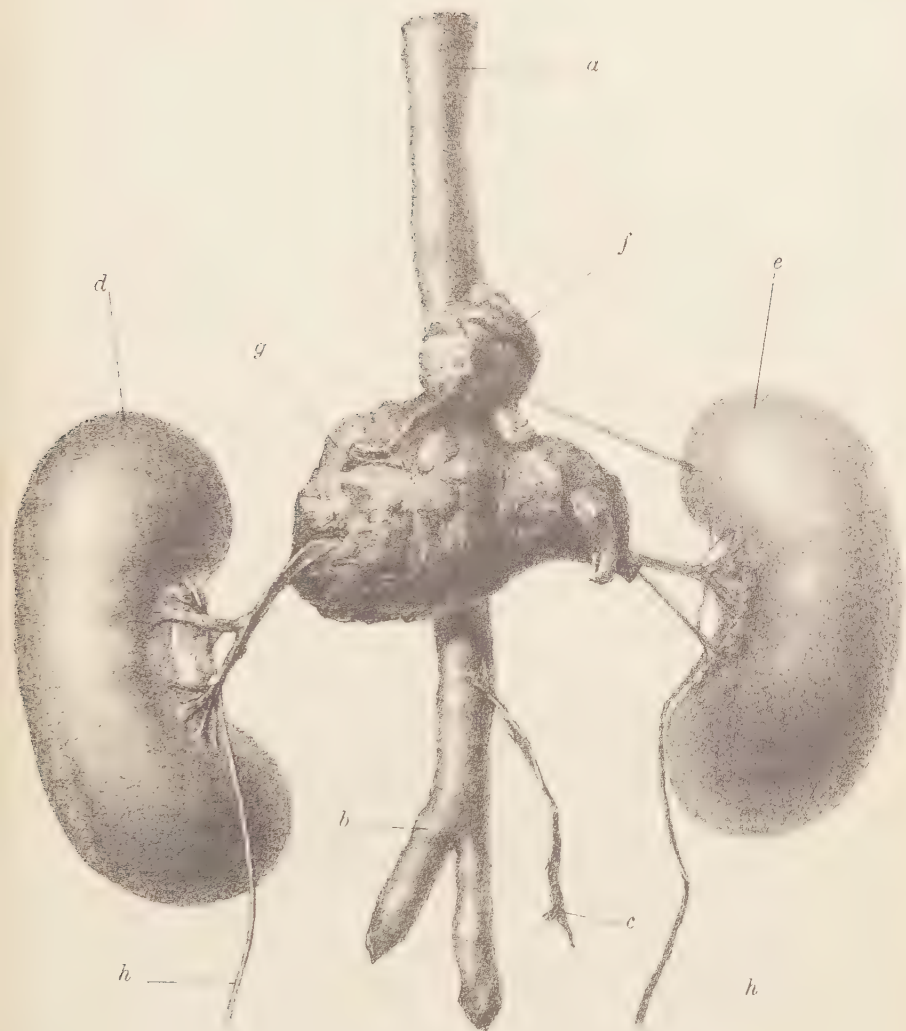


FIG. 267.

Aneurism of the Abdominal Aorta, involving the origins of the celiac axis, superior mesenteric and renal arteries. (Smaller than the actual specimen—a dried preparation. Size of drawing, $5\frac{1}{4} \times 4\frac{1}{2}$ in.; size of specimen, $8\frac{1}{2} \times 7\frac{1}{4}$ in.).

a, descending thoracic aorta; b, abdominal aorta at its bifurcation; c, inferior mesenteric artery; d, the right, and e, the left kidney; f, the upper sac of the aneurism, which involves the origin of the celiac axis; g, the lower sac from which the main branches of the renal arteries spring; h, h, the ureters.

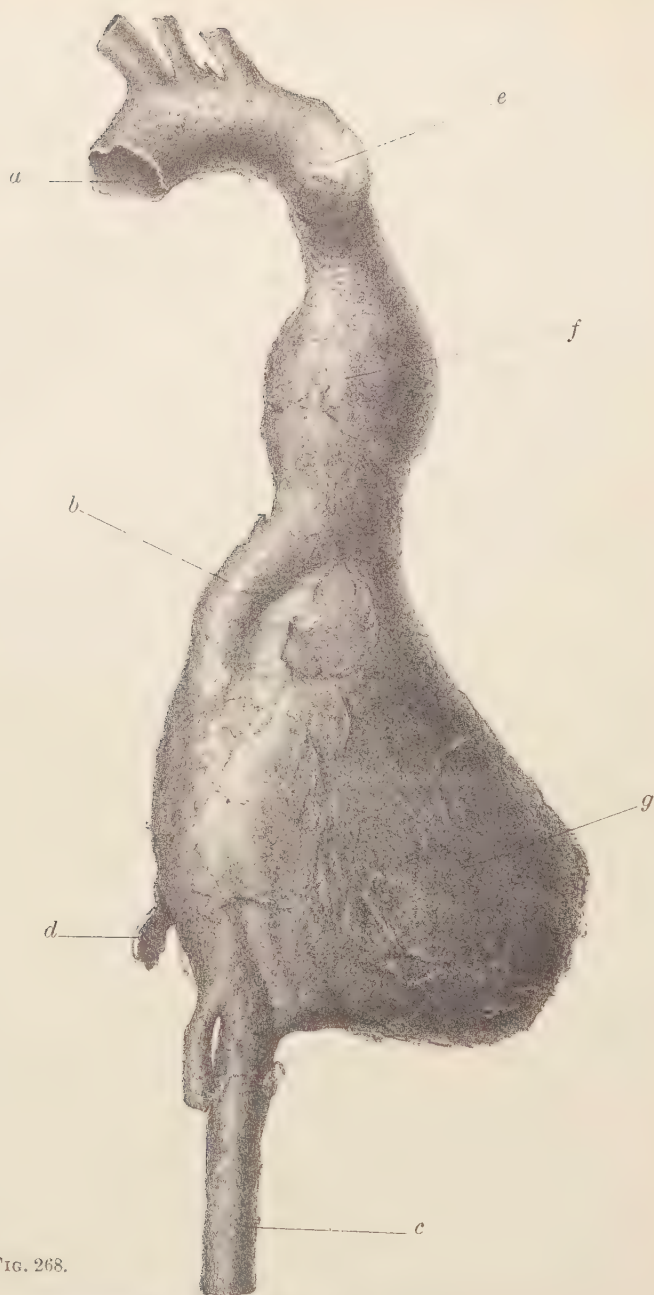


FIG. 268.

*Aneurism involving the descending thoracic and the upper part of the Abdominal Aorta. (Front view.)
Smaller than the dried preparation. Size of drawing, $6\frac{1}{2} \times 2\frac{1}{2}$ in.; size of specimen, $13\frac{1}{2} \times 5\frac{1}{4}$ in.*

a, Termination of the ascending portion of the aortic arch; b, a portion of the descending thoracic aorta situated between the aneurismal sacs f and g; c, abdominal aorta below the aneurism; d, the diaphragm; e, small commencing aneurism at the junction of the transverse with the descending portions of the arch; f, globular aneurismal sac, the size of a hen's egg, involving the descending thoracic aorta; g, large heart-shaped aneurism, springing from the descending thoracic and the abdominal aortæ.



FIG. 269.

*Aneurism involving the descending thoracic and the upper part of the Abdominal Aorta. (Back view.)
Smaller than the dried preparation. Size of drawing, $6\frac{1}{2} \times 2\frac{1}{2}$ in.; size of specimen, $13\frac{1}{2} \times 5\frac{1}{4}$ in.*

a, termination of the ascending portion of the aortic arch; b, abdominal aorta; c, the diaphragm; d, globular aneurismal sac, involving the descending thoracic aorta; e, large heart-shaped sac, springing from the descending thoracic and the abdominal aortæ; f, point at which the upper sac was in contact with the spinal column; g, point at which the lower sac was in contact with the spinal column. A septum separates the two sacs internally. The spinal column corresponding to the points f and g was eroded.

FIG. 272.

FIG. 271.

FIG. 271.—Longitudinal section through the wall of the aorta and the adjacent wall of an aneurism springing from it. (About 10 diameters.)

a, inner coat of aorta; *a'*, *a'*, detached portions of the inner coat; *b*, middle coat of aorta; at *b'* the inner coat is detached; *c*, the wall of the aorta at the orifice of the sac; *d*, *d*, greatly thickened inner coat, lining the interior of the aneurismal sac; *e*, *e*, processes on the middle coat of the aneurismal wall, it has almost entirely disappeared; *f*, *f*, obliterated blood vessels in the cellular tissue (*g*) which lies between the aorta and the aneurism, i.e. in the outer coat of the aorta—for the aneurism and the sac wall are in close contact here.

FIG. 272.—Diagram to show the relationship of the aorta and the aneurism. The letter *a*, which is placed in the sac of the aneurism, points to the part of the sac wall represented in fig. 271; *b*, is placed in the aorta.

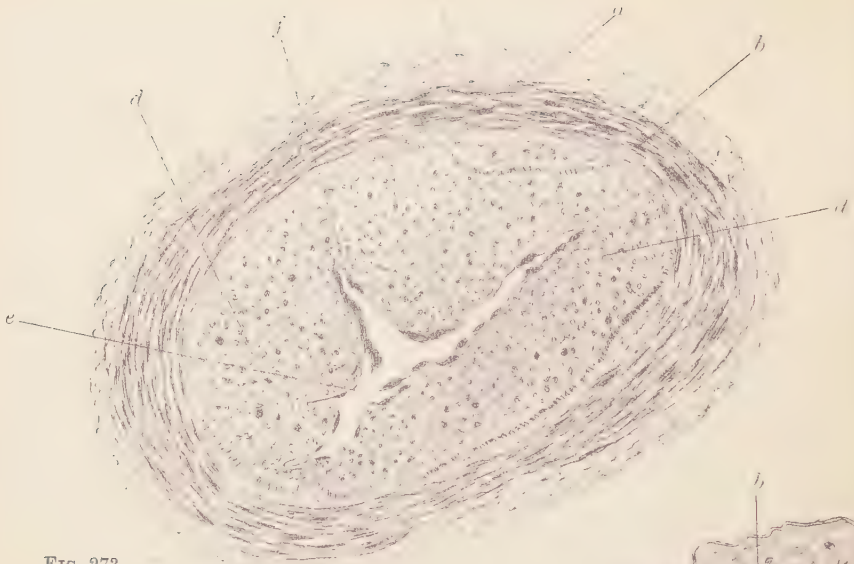


FIG. 273.

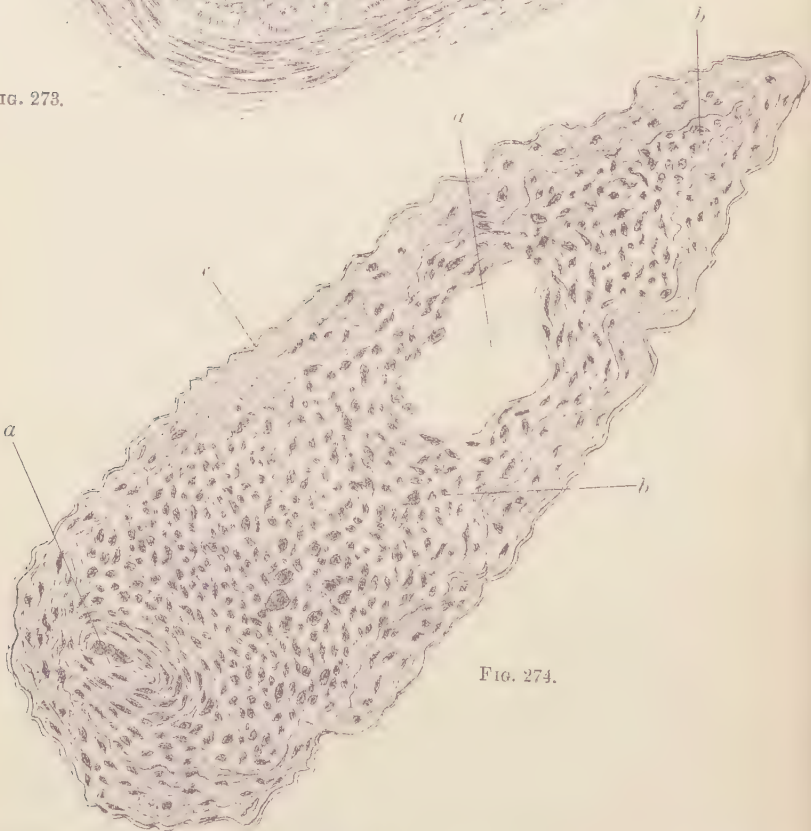


FIG. 274.

FIG. 273.—Transverse section through an artery affected with endarteritis obliterans—the vessel to which the letter *a* in fig. 245 points. (Magnified about 180 diameters.)

a outer, and *b*, middle coats; *c*, elastic lamina; *d*, *d*, tissue filling up the vessel; *e*, free lumen which still remains; *f*, large cells lining the interior of the diseased vessel.

FIG. 274.—Transverse section through an artery in endarteritis obliterans—the vessel to which the letter *f*' in fig. 271 points. (Magnified about 250 diameters.)

a, *a'*, unobliterated spaces in the centre of the vessel; *b*, *b*, cellular elements filling up the lumen of the vessel; *c*, elastic lamina.

middle or muscular coat is by far the most important factor ; and as far as my observation goes, it fully confirms that of Cornil and Ranvier, who state, that in all spontaneous aneurismal sacs the middle coat has either partially or totally disappeared. In many cases, the degeneration of the media is secondary to the lesion of the interna. In others, the disease of the middle coat is primary, and is, I think, in some cases due to an obliterative affection (endarteritis obliterans) of the nutrient vessels in the aortic wall. (See fig. 273.) If this opinion be correct, it affords an explanation of the manner in which syphilis may act, in the production of aneurism. Acute ulceration of the inner coat, which is met with at the base of the aorta in some cases of ulcerative endocarditis, is another cause of aortic aneurism (see fig. 168, in which a small local dilatation is present just above the diseased valves) ; but in cases of this description, the patient seldom survives a sufficient length of time to allow the aneurism to attain any but very small dimensions.

It follows therefore that, anything which produces arterial degeneration, predisposes to the formation of aneurism : syphilis, alcoholic excesses, increased aortic blood pressure (whether it occurs during the whole period of the cardiac cycle, as in cirrhosis of the kidney, or only during the cardiac systole, as in aortic regurgitation), strain, a laborious occupation, gout, and rheumatism, are the chief causes of the condition. Increased blood pressure tends not only to produce arterial degeneration, and so predisposes to the production of aneurism, but it also acts directly by causing the degenerated and weakened part to give way. Sudden increase of the blood pressure (such as is produced by violent muscular exertion or sudden effort) is more likely to produce an aneurism than the continued high blood pressure, for example, which is seen in Bright's disease. And this is more especially the case when, in addition to the violent muscular exertion, which produces a sudden increase in the arterial blood pressure, the arterial circulation is impeded, as, for instance, it is apt to be in soldiers by tight and badly fitting accoutrements. In rare cases, traumatic injuries, such as blows on

the chest, falls, etc., seem to be the direct cause of the condition. In such cases the artery which gives way was probably already diseased; and in the case of a fall, at all events, it is more frequently the sudden effort which the patient makes to save himself, and the consequent sudden increase in the arterial blood pressure, which produces the aneurism, rather than any direct injury to the arterial coats.

Thoracic aneurisms occur more frequently between the ages of thirty and forty-five than at any other period of life, for it is at this time that their two great causes—arterial degeneration and strain—are most often met with in combination. The condition is extremely rare before the age of twenty, and is seldom seen before thirty; for although young persons are frequently exposed to strain, their arteries are sound, and therefore able to bear a sudden increase of the blood pressure. After the age of fifty the condition becomes less common; for although the tendency to atheroma increases with the age of the patient, the circulation in old people is seldom put upon the stretch, and the degenerated and weakened aorta is therefore equal to the strain to which it is exposed.

Occupation necessarily exerts a very important influence upon the production of aneurism. Soldiers (who frequently contract syphilis, who are often given to alcoholic excesses, and whose arterial pressure is apt to be suddenly raised to a very high point in consequence of the powerful muscular exertions and sudden efforts which they have to make, and the tight-fitting accoutrements which they have to wear) are more liable to aneurism than any other class of the community. Speaking generally, it may be stated that all laborious occupations predispose to aneurism. Prostitutes suffer from aneurism much more frequently than other females. In exceptional instances, of which I have previously quoted an example (see page 86), the tendency to thoracic aneurism seems to be hereditary.

Any part of the thoracic aorta, from its commencement just above the aortic orifice (sinuses of Valsalva, see fig. 270), to its termination beneath the pillars of the diaphragm, may

be affected; but the ascending portion, and the junction of the ascending and transverse portions of the aortic arch, are the parts of the vessel which are most frequently involved.¹ These parts of the aorta, especially the ascending portion of the aortic arch, receive, as it were, the full force of the blood current, as it is discharged from the left ventricle, and are therefore more exposed to strain than other parts.

On microscopical examination, the wall of an aneurism is, in most cases, found to be composed of the inner and outer coats of the aorta, modified by inflammation or degeneration, the middle having totally or partially disappeared. At the bottom of the sac the middle coat is not seen, but at the neck of the sac (see fig. 271) it is generally present; it tapers away, and finally disappears altogether, as the aneurismal wall passes away from the junction of the sac with the aorta. The thickened and altered inner and middle coats then come into direct contact. The blood vessels in the outer coat are often completely or partially obliterated by endarteritis deformans. Layers of fibrine, in which white blood corpuscles are often embedded, are usually found lining the inner surface of the sac, and seem in some preparations to be undergoing a process of imperfect organisation. The degenerated and thickened inner coat often presents fatty and atheromatous changes, or is infiltrated with calcareous deposits.

Clinical History.—In considering the symptoms and physical signs of aneurisms of the thoracic aorta, it is important to remember:—

Firstly, That a thoracic aneurism is a pulsating tumour, which is placed in a cavity, the walls of which are, for the most part, rigid; and that, as the tumour increases, it necessarily

¹ The term *true* used formerly to be applied to those aneurisms, the walls of which are composed of all the three arterial tunics (intima, media, and adventitia); we now know that the media is almost invariably absent at the seat of greatest dilatation, *i.e.* at the bottom of the sac. The term *true*, as applied in the text, includes all those aneurisms in which any of the proper walls of the artery still remain as a continuous wall over the sac, in opposition to the term *false*, in which all the three coats have ruptured, and the contents of the original (*true*) sac have become extravasated into the surrounding tissues.

pushes aside and presses upon the surrounding organs and parts.

Secondly, That the results of this pressure are partly *mechanical* (displacement of the solid organs and parts, collapse or constriction of hollow organs, such as the lung or œsophagus) and partly *vital* (irritation, inflammation, and ultimately destruction and absorption of tissue).

Thirdly, That the size of the sac is constantly undergoing changes and variations, in accordance with alterations of the blood pressure; and that the direction, in which the aneurism extends, is apt to undergo variations, in consequence of the fact, that first one part of the sac wall, and then another, yields before the internal pressure. In consequence of these variations, which may occur with considerable rapidity, a structure which to-day is seriously pressed upon, may to-morrow be much less seriously implicated. Rapid modifications in the pressure symptoms are not unfrequently, therefore, observed. The exact nature of the pressure-effects varies with:—

(1) The position and size of the sac; in other words, with the particular organs and parts which are exposed to the pressure.

(2) The static condition of the sac, the degree of pressure which is being exercised (*i.e.* the internal blood-pressure), and the particular direction in which the sac is extending.

Cases of thoracic aneurism may, for clinical purposes, be divided into three great groups.

In the *first* group the aneurism is entirely latent. In cases of this description, there are no symptoms, and there *may* be no physical signs. (It is quite possible, however, that in some cases physical evidence of aortic or cardiac disease would be detected, if the physician had occasion to make a careful examination of the chest.) In the cases included in this group, the aneurism is deeply situated, usually of small size, and is not exerting injurious pressure on any of the surrounding parts.

In a *second* group of cases, there are very distinct symptoms and signs of intra-thoracic pressure, but it is extremely

difficult—it may be impossible—to determine whether the pressure is due to an aneurismal tumour or a solid intra-thoracic growth. In these cases the aneurism is deeply situated, and the physical signs are obscure.

In a *third* group, the physical signs of aneurism are very distinct, and there usually are very distinct pressure symptoms. In such cases in which the aneurism is, as a rule, superficial, and often of large size, the diagnosis is easy or self-evident.

Let us now consider the exact character of the symptoms and physical signs, which may be present in cases of aneurism of the thoracic aorta, in detail.

Symptoms.—The most important symptoms of aneurism of the thoracic aorta are the pressure symptoms. In many cases there is little or no derangement of nutrition, little disturbance of the general health. It is impossible to insist too strongly upon this important fact, which is in many cases of great diagnostic value. Cases are again and again met with in which a patient who is muscular and well nourished, and who presents all the appearances of robust—it may be of plethoric—health, is found on examination to be the subject of aneurism. Thoracic aneurism is, in fact, one of the few conditions in which a disease, which may at any moment prove fatal, may fail to produce any external manifestation, even to the eye of the most accomplished clinical observer. It must not, however, be supposed that in all cases of thoracic aneurism the condition of the general health and the physiognomy of the patient are unaltered. In many cases the countenance wears a worn expression, suggestive of internal suffering or of repeated attacks of pain. In some, the patient looks exhausted, and is more or less—but seldom profoundly—emaciated, the exhaustion being generally due to long continued pain or sleeplessness; while the emaciation is caused by pressure on the œsophagus or thoracic duct, or by some complication. In those cases in which the sac is pressing upon the superior cava, the face may be swollen and the lips livid. Sometimes the physiognomy is suggestive of a cardiac complication.

The more important *pressure symptoms* are as follows :—

Pain.—Of all the symptoms of aneurism of the thoracic aorta this is the most frequent. In many cases the pain is distinctly intra-thoracic, and is then, I believe, generally due to irritation of the fine sensory nerve filaments of the aorta itself, or of the surrounding organs on which the sac is pressing. In these cases the pain is usually more or less intermittent, but it is not sharp, shooting and lancinating in character, and is not so distinctly paroxysmal as the pain due to pressure on a large nerve trunk. In other cases the pain is more constant, and is referred to some part of the chest wall, being in many cases very localised, and presenting a dull boring character. Pain of this description is generally due to pressure on the chest wall, and is typified by pain in the back, which results from the pressure of an aneurismal sac upon the spinal column, and from erosion of the vertebræ. In a few cases in which the pain is distinctly angina-like in character, the aneurism usually involves the root of the aorta, and the pain is probably due to the pressure on the cardiac plexus. In others again, the pain radiates very distinctly in the area of distribution of some large nerve trunk, shooting, for example, round the chest along the course of an intercostal nerve, or down the arm in the area of distribution of the nerves of the brachial plexus. In cases of this nature, the pain is markedly paroxysmal and lancinating in character. It may be attended with some anæsthesia of the skin and loss of motor power; it presents, in short, the ordinary characters of pain, which is due to direct irritation of a common sensory motor nerve trunk.

Dyspnœa, cough, and alterations in the voice, are frequently observed in cases of thoracic aneurism, and may be due to a variety of causes. A large intra-thoracic aneurism necessarily interferes with the expansion of the lungs and the free play of the heart, and by its mere size acts as a mechanical cause of shortness of breath; but dyspnœa—more especially the severe forms of dyspnœa—cough, and other respiratory symptoms, are generally due to local irritation caused by the pressure of the sac on some part of the respiratory apparatus.

Respiratory symptoms (dyspnœa, cough, etc.) may also, of course, be due to associated disease of the lungs and heart.

The pressure of the aneurismal sac on the lung tissue very frequently produces local pleurisy, in consequence of which, adhesions are formed which bind the lung to the thoracic wall. When the lung tissue itself is pressed upon and irritated, cough, expectoration (mucous, muco-purulent or bloody in character), and shortness of breath (seldom amounting to dyspnœa) are usually present. If the symptoms and signs of aneurism are absent or indistinct, the observer may perhaps suppose that he is dealing with a case of phthisis. An aneurism which is pressing upon the lung may ultimately burst into the lung tissue; the fatal rupture is in some cases preceded by blood-tinged or rusty expectoration, a symptom which should always excite grave apprehension when it occurs in the course of this affection (*i.e.* of aneurism).

Pressure on a main bronchus usually produces considerable dyspnœa, which in some cases is paroxysmal; an extremely irritable and troublesome cough, which is generally attended with a copious, thin, watery expectoration, is often present. A wheezing sound can sometimes be heard when the stethoscope is placed over the position of the compressed bronchus. Pressure on a main bronchus prevents the free entrance of the air into the lung, the respiratory sounds are consequently indistinct on the affected side, but this point will afterwards be considered more in detail.

Pressure on the trachea usually gives rise to great distress and difficulty in breathing, and may produce the most aggravated form of orthopnœa. Dr Bristowe is of opinion that pressure on the trachea is the cause of the sudden attacks of paroxysmal dyspnœa, which occur in the course of some cases of thoracic aneurism, and which not unfrequently prove fatal. Aneurisms of the transverse portion of the aortic arch are most likely to produce this form of dyspnœa (which is by many observers thought to be the result of pressure on the recurrent laryngeal nerve), and therefore to cause laryngeal paralysis or spasm. The suddenness of the attack, if we adopt Dr Bristowe's view, may be explained by

supposing either that there is a rapid increase in the pressure, with consequent rapid tracheal obstruction, or that the narrow slit-like aperture in the trachea, which is the result of the compression, becomes suddenly obstructed by an accumulation of mucus. Dyspnœa, the result of tracheal compression, is usually accompanied by a well-marked stridor. The observer at once sees, on looking at the patient, that there is difficulty in getting a sufficient quantity of air into the chest. There is often, in addition to the dyspnœa and stridor, a troublesome and violent cough. It is sometimes possible, by altering the position of the patient, to remove, in part at least, the pressure from the trachea, and so to relieve the dyspnœa.

Pressure on the recurrent laryngeal nerve is another very important cause of difficulty in breathing and cough. The dyspnœa is apt to occur in paroxysms, and in some cases seems to be due to paralysis, in other cases to spasm of the glottis. Pressure on a nerve trunk gives rise either to irritation or destruction; irritation of a motor nerve produces spasm, while destruction causes paralysis. Now the left recurrent laryngeal nerve is the motor nerve which supplies almost all the muscles of the left side of the larynx; consequently when it is irritated by the pressure of an aneurism or any other cause, spasmodic contraction of the left vocal cord is produced. When, again, the pressure is more considerable, and the nerve fibres are destroyed, paralysis of the left vocal cord will result. These changes can, of course, be recognised with the laryngoscope. In quite exceptional cases pressure on the left recurrent has been attended with bilateral laryngeal paralysis. A hard, dry, barking, clanging cough, which is in some cases paroxysmal, is also produced by pressure on the recurrent laryngeal nerve. The voice, too, is usually hoarse, husky, or whispering; in some cases complete aphonia is observed.

Paroxysmal attacks of dyspnœa, resembling asthma, seem in some cases to be due to irritation of the pulmonary branches of the vagus.

Dysphagiā is present in many cases of intra-thoracic

aneurism, and is generally due to compression and flattening of the œsophagus, but in some cases it seems to be caused by spasms of the muscular fibres of the œsophagus. The difficulty in swallowing is rarely constant, but is apt to vary from time to time. It is rarely so complete as the dysphagia which results from an organic stricture. It is important to emphasise the fact, that a bougie should never be passed in cases of this description, lest the pressure of the instrument should rupture the aneurismal sac and produce fatal hæmorrhage. In all cases of dysphagia, therefore, before passing an œsophageal bougie, the physician should make a careful examination of the chest, and should satisfy himself that the difficulty in swallowing does not depend upon the presence of an aneurism.

Engorgement of the superior cava and its branches, and œdema of the subcutaneous cellular tissue of the head, neck, upper extremities and upper part of the thoracic wall, are seen in some cases of thoracic aneurism, and are due to the pressure of the sac upon the superior cava. The hard, brawny swelling of the subcutaneous tissues at the root of the neck, to which the term ‘collar of flesh’ has been applied, is sometimes observed.

Emaciation may result from pressure on the thoracic duct; it is, however, more frequently due to derangement of the functions of the stomach or to obstruction of the œsophagus.

Hiccough and paralysis of one-half of the diaphragm may be caused by pressure on the phrenic nerve.

Dyspepsia is sometimes caused by pressure on the vagus.

Physical signs.—The physical signs, which are present in cases of thoracic aneurism, vary considerably in different cases. They may be divided into two groups, viz. :—

1st. *The primary or direct physical signs*, i.e. the physical signs which are directly derived from the aneurism itself.

2d. *The secondary or indirect physical signs*, i.e. the physical signs derived from the altered condition of the organs or parts on which the aneurismal sac presses, and from the

secondary alterations in the circulation behind and in front of the aneurismal dilatation.

Primary physical signs.

The primary physical signs depend more particularly upon :—

- (1) The size of the sac.
- (2) The position of the sac, more especially its relationship to the chest wall.

It is of the greatest importance to remember that the condition of the lungs exerts a most important influence upon the nature of the physical signs ; when, for example, the lungs are emphysematous, it may be difficult or impossible to detect an aneurism, even of large size, by physical examination : when, on the contrary, the anterior margins of the lungs are retracted and the aneurismal sac comes in contact with the chest wall, the physical signs are unusually distinct.

Inspection.—(a) When the aneurism is of small size, when it is deeply situated and separated from the chest wall by the lung tissue, inspection of the chest yields no information.¹

(b) When the sac is in contact with the chest wall, but when the chest wall is still intact, slight prominence and pulsation can usually be seen on careful examination.¹

(c) When the aneurism has eroded the chest wall, an external pulsating tumour is apparent. The size of the tumour varies, of course, in different cases ; it not unfrequently is as large as the closed fist, and in some cases has been known to attain the size of a child's head.

The position of the pulsating prominence depends upon the part of the artery which is affected, and the exact spot at which the sac 'points.' Aneurisms of the ascending aorta, for example, usually perforate the chest in the neighbourhood of the second or third right interspace ; aneurisms of the descending portion of the aortic arch, or of the descending

¹ The exact manner in which the chest is to be inspected in order to detect slight degrees of pulsation and prominence, has been previously described. (See p. 228.)

thoracic aorta, generally 'point' on the posterior or lateral wall of the left chest, but this will afterwards be considered more in detail. The base of the prominence is usually much broader than the apex. The skin over the surface of the prominence may be quite natural, in other cases it is tense, shining, red or bluish in colour.

Palpation.—(a) When the aneurism is of small size, or when it is deeply situated and separated from the chest wall by lung tissue, palpation of the chest wall over the sac may yield no information. In some cases deep pulsation can be felt by the bimanual method of palpation previously described (see p. 229); in others, more particularly when the transverse portion of the aortic arch is affected, pulsation can be felt in the supra-sternal notch.

(b) When the aneurismal sac is in direct contact with the chest wall, but when the chest wall is not perforated, a systolic impulse, and, in some cases, a diastolic shock can be felt when the hand is placed lightly over the position of the sac. A systolic thrill can also sometimes be perceived. (This sign is more frequently present in cases of general dilatation than in cases of saccular aneurism.)

(c) When the sac has perforated the chest wall, the pulsating prominence is in some cases soft, fluid, and fluctuating; in others (*i.e.* in those cases in which the external sac contains much laminated clot), it may feel firm and solid. A thin, external sac, must of course be handled with great care. The skin covering the sac, and the structures forming the base of the external sac are often extremely tender to the touch.

Percussion.—(a) When the aneurism is of small size or deeply situated and separated from the chest wall by lung tissue, more especially when the lungs are emphysematous, percussion may yield negative results.

(b) When the sac is in contact with the chest wall there is dulness on percussion and a feeling of increased resistance over the sac. The position of the dulness depends upon the portion of the artery which happens to be affected, and

the part of the chest wall with which the sac is in contact. The dulness is not, of course, an exact indication of the size of the sac, for some part of the tumour is always separated from the chest wall by resonant lung tissue. When the chest wall is tender to the touch, or when there is a thin-walled external sac, the percussion must be lightly performed. Impaired resonance may be detected in many cases in which the sac is not in direct contact with the chest wall.

Auscultation.—In order to comprehend the exact significance of the sounds, which *may* be heard over an aneurismal sac, it is essential to remember:—

(1) That the sounds and murmurs which are generated within the heart, more especially the aortic second sound and aortic valvular murmurs, are propagated into the aorta, and may therefore be heard over the position of the aneurism.

(2) That in dilated or aneurismal conditions of the aorta, the aortic second sound is very frequently accentuated; and that this accentuated second sound (which is produced, be it remembered, at the aortic orifice and not in the sac itself) may be heard over the aneurismal tumour.

(3) That in the majority of cases of thoracic aneurisms, no new sounds are generated within the sac itself.

(4) That in the minority of cases, a murmur is generated within the dilated and atheromatous or aneurismal aorta; and that this murmur is almost invariably systolic. In very rare and exceptional cases, a diastolic murmur (generated within the sac itself) has been heard.

Now when the aneurismal sac is small or deeply situated, auscultation may afford no direct evidence of its presence. A murmur or an accentuated second sound may, it is true, be heard when the stethoscope is placed over the surface of the chest overlying the sac, but the murmur or accentuated second sound is not louder (nearer to the ear) than it would be if the aneurism were not present. Accentuation of the aortic second sound is suggestive of the presence of an aneurism, but in the absence of other symptoms and signs it is of no great value, for it may depend upon a variety of

other conditions. In other words, an accentuated second sound is not *direct* evidence of the presence of an aneurismal tumour. An accentuated second sound, when heard over a circumscribed dull area in the course of the aorta, is however, very suggestive of the presence of an aneurism.

When an aneurismal sac approaches the surface of the chest, the sounds, which are generated at the aortic valve or in the aneurismal sac itself, are of course brought nearer to the ear. When, therefore, sounds or murmurs are heard over a part of the chest wall, at which, in health, the aortic cardiac sounds are feebly or indistinctly heard, the presence of an aneurism is strongly suggested, but by no means proved; for anything which facilitates the conduction of a sound between the aorta on the one hand and the chest wall on the other (such, for example, as a solid intra-thoracic growth), may produce the like result. The exact value, which is to be attached to evidence of this description, depends upon:—

(1) *The character of the sounds.*—An accentuated second sound, a double murmur or a diastolic murmur, would, under such circumstances (*i.e.* when heard over a part of the chest at which under normal circumstances the aortic or cardiac sounds are indistinct) be strongly suggestive of an aneurism. A systolic murmur, accompanied by a well-marked or accentuated second sound would also be in favour of an aneurism. But a systolic murmur alone, *i.e.* unaccompanied by a well-marked or accentuated second sound, is of little or no value; in fact, other things being equal, it would be more likely to be produced by a solid intra-thoracic tumour compressing the aorta, than by a dilated or aneurismal condition of the vessel. A systolic murmur heard in the back, over the position of the descending thoracic aorta is, however, of great importance from a diagnostic point of view.

(2) *The position at which the sounds are heard.*—When the sounds or murmurs are heard over the course of the aorta, an aneurism is suggested; when they are heard away from the course of the aorta, some other cause of increased conduction is probable.

(3) *The associated symptoms and physical signs.*—In many

doubtful cases the significance of the sounds depends entirely upon the associated conditions. In those cases, for example, in which a solid tumour is in contact with the aorta, and in which the aortic sounds are conducted with increased facility to the chest wall, the conduction of the lung sounds is also facilitated; bronchial breathing, in such instances, may often be heard over the position of the tumour.

This point will be further considered in treating of the diagnosis.

When the aneurismal sac is in direct contact with the chest wall, or when it has perforated and produced an external tumour, the shock of the aneurismal impulse can often be distinctly *felt* when the ear is placed on the stethoscope. A jog or shock of this description, which I am in the habit of terming a shock-sound,¹ is very characteristic of a large thin-walled superficial sac. In most cases, the shock is systolic; in some a diastolic shock, which is even more characteristic and distinctive, may also be perceived. When the aortic second sound is accentuated or when a murmur is produced either at the aortic orifice or in the sac itself, these modifications may of course be heard when the stethoscope is placed over a superficial sac which is in contact with, or has perforated the chest wall.

Secondary physical signs.

The secondary physical signs are, as I have previously pointed out, of two kinds, viz. :—

(1) Those which are produced by secondary changes in the heart and circulation, behind and in front of the aneurismal tumour respectively.

(2) Those which are derived from the altered condition of the organs and parts (other than the heart, arteries, and veins) on which the aneurismal sac presses.

¹ The term shock-sound is not perhaps very elegant, but it is expressive, and conveys to my mind, at least, the fact that the impression which is perceived by the ear, is chiefly that of shock, accompanied by a faint and almost toneless sound.

Physical signs due to alterations in the heart and circulation.

A. Backward effects.—An aneurism of the thoracic aorta does not necessarily produce any secondary alteration in the condition of the heart. In many cases, however, cardiac alterations are present. These alterations may be the direct result of the aneurism, but in many cases they are merely associated conditions, the aneurism of the aorta and the cardiac lesion being the results of a common cause, as, for instance, atheroma.

Hypertrophy of the heart.—In some cases the heart is hypertrophied; the enlargement may be due to the increased difficulty which the left ventricle meets with, in forcing the blood through the aneurismal dilatation, but is generally the result of some other condition, such as associated valvular disease, general atheroma, etc. An aneurism of the thoracic aorta does not necessarily, therefore, produce hypertrophy of the left ventricle, and this is more particularly the case when the aneurism is saccular. (In cases of general dilatation of the aorta, the left ventricle is usually hypertrophied.)

Displacement of the heart is very frequently observed. The displacement is, as a rule, downwards, or downwards and to the left. In exceptional cases (as, for instance, in that represented in figure 268) the heart is displaced forwards.

Incompetence of the aortic valve is very often met with. In some cases, the incompetence is the direct result of the aneurism, as, for instance, in those cases in which the base of the aorta is affected and relative incompetence of the aortic valve produced, the aortic segments themselves being healthy (see fig. 270). In most cases the incompetence is only an associated condition, and is not the direct result of the aneurism, the two conditions (the aneurism and the aortic incompetence) being due to a common cause, *e.g.* atheroma or syphilitic disease of the aorta. In a few cases, the aortic incompetence seems to be the cause of the aneurism; but this point has been previously mentioned under the head of ætiology.

Pericarditis, adherent pericardium.—Aneurisms of the thoracic aorta, more especially of the intra-pericardial portion of the ascending arch, very frequently produce pericarditis.

My observations are not sufficiently large to enable me to draw any exact statistical conclusions on this point, but I have been very forcibly struck with the large proportion of cases in which the pericardium is found to be adherent after death. The symptoms and signs of pericarditis are met with in the course of some aneurisms during life; and so satisfied am I of the frequency with which pericarditis is produced by the pressure of an aneurismal sac, that in any case of non-rheumatic pericarditis occurring after the age of forty, in which the cause of the pericarditis is obscure, I strongly suspect the presence of an aneurism. (In many cases in which the symptoms of angina pectoris are associated with the physical signs of pericarditis, and in which the cause of the pericardial inflammation is obscure, as for example in the case recorded on page 310, the primary lesion is, I believe, an aneurism of the root of the aorta.)

Compression of the pulmonary artery.—An aneurism of the root of the aorta may compress the pulmonary artery and produce a systolic pulmonary murmur. It seldom happens that the compression is sufficiently great to produce much obstruction to the blood flow, and (consequent) venous engorgement.

Compression of the left auricle is not unfrequently produced by an aneurism of the root of the aorta, but the compression is seldom sufficiently great to impede the blood flow through the pulmonary circuit.

Compression of the inferior cava, or of the left innominate vein is not uncommon, and is of course most apt to arise when the ascending or transverse portions of the aortic arch are affected. As the result of the compression, the return current of blood from the head and neck, upper extremities, and upper surface of the chest (through the internal mammary veins) is interfered with; the external jugular and other veins, which discharge into the vena cava, become distended, and subcutaneous dropsy, swelling of the face and the other conditions, which have been previously described, ultimately may occur. In some cases, the pressure of the aneurism produces inflammation of the venous coats; the vein may then become plugged, and may be felt as a dense firm cord, which, in

some cases (*i.e.* in the earlier stages of the condition) is tender to the touch. In a few cases, the aneurism ruptures with the vein, and an arterio-venous murmur is established. It is important to remember, as I shall afterwards point out in speaking of the diagnosis, that aneurismal tumours are less likely to produce obstruction of the venous return than solid intra-thoracic growths.

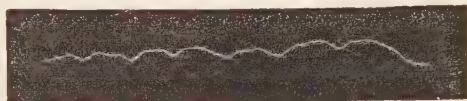
B. *Forward effects.*—In many cases of intra-thoracic aneurism (more especially when the aneurismal sac is situated between the heart and the origin of the great blood-vessels which arise from the transverse portion of the aortic arch, or when these vessels are themselves implicated or pressed upon by the aneurismal sac), the pulse in the arteries on the peripheral side of the sac is materially altered. These alterations, which constitute one of the most important secondary or indirect physical signs of the disease, are in some cases due to the fact that the blood wave, as it passes through the sac of the aneurism, is modified; in others to the circumstance, that the aneurismal sac presses upon and obstructs one or other of the vessels (innominate, left common carotid, or left subclavian) which spring from the aortic arch. Alterations in the peripheral arteries are usually best observed in the radial arteries; but the condition of the carotid and femoral pulse should also be noted.

The pulse wave (or rather the period of maximum impulse of the pulse wave) is retarded, and the curves of the pulse wave are, as it were, flattened out or effaced. In well-marked cases, the up-stroke of the sphygmographic tracing is sloping, the apex rounded, and the secondary curves more or less or entirely obliterated. These alterations are best marked when the aneurismal dilatation is globular, and when the walls of the sac are elastic. (See figs. 275, 276, 277, and 278.)

The retardation of the pulse is best appreciated by the finger; the obliteration of the pulse curves by means of the sphygmograph.

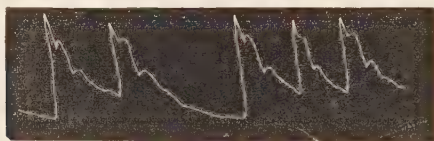
Differences in the pulse tracings from the two wrists are not observed in all aneurisms. When the arch of the aorta, below the origin of the innominate is involved, the pulse wave

in the two wrists is the same,¹ though the sphygmographic tracing on each side may be modified, each pulse wave being



Pressure 3 oz.

FIG. 275.—*Aneurism of Left Axillary Artery (left radial tracing)*.—L. G., æt. 63, admitted to the Newcastle Infirmary 7th March 1878, with a large aneurism of the left axillary artery. The apex is rounded; all the curves are obliterated.



Pressure 3 oz.

FIG. 276.—*Aneurism of Left Axillary Artery (right radial)*.—Right radial tracing for the same patient. The pulse is intermittent, but all the curves are well marked.



FIG. 277.—(Left radial). Pressure 3 oz. FIG. 278.—(Right radial). Pressure $3\frac{1}{2}$ oz.

FIGS. 277 and 278.—*Aneurism of Left Subclavian*.—J. M., æt. 50, admitted to Newcastle Infirmary 5th September 1878; all the waves in the left tracing are obliterated.

affected, *quoad* its curves in a like degree. (See figs. 279 and 280.) When the aneurismal sac involves the innominate, the left subclavian being unaffected, the right radial pulse is modified, while the left is normal. When the sac is situated between the origin of the innominate and left subclavian, or when the left subclavian is implicated (the circulation through the innominate being uninterfered with), the right radial pulse is normal but the left may be modified. (Further details

¹ An exception to this statement occurs, when an aneurism of the ascending portion of the aortic arch presses upon or displaces the innominate artery so as to interfere with the free passage of the blood through that vessel, without pressing upon or interfering with the passage of the blood current through the left subclavian artery.

have previously been given in treating of the pulse and sphygmograph, see p. 283.)

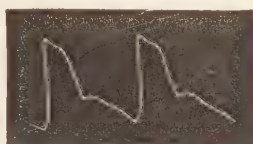
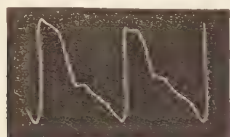


FIG. 279.—(Right radial.) Pressure $\frac{1}{2}$ oz. FIG. 280.—(Left radial.) Pressure $\frac{3}{4}$ oz.

FIGS. 279 and 280.—*Aneurism of Ascending portion of Aortic Arch.*—J. D., æt. 50, admitted to Newcastle Infirmary suffering from a large aneurism of the ascending thoracic aorta and atheroma. There is no important difference between the two pulses.

Embolic symptoms sometimes arise from portions of fibrine being washed away from the aneurismal sac and carried to some distant vessel. This accident is of less frequent occurrence than might theoretically be supposed. The exact nature of the symptoms depends, of course, upon the vessel which happens to be obstructed and the function of the part which it supplies.

Physical signs due to altered condition of organs and parts other than the heart, arteries, and veins.

The secondary physical signs, which result from the pressure of the aneurismal sac upon the organs and parts which surround it, are both numerous and important. Some of them have already been incidentally mentioned in speaking of the '*pressure symptoms*.'

When the aneurismal sac presses on the *right bronchus*, and interferes with the free entrance of air into the right lung, the respiratory murmur is fainter on the right side than on the left; the percussion note over the right side of the chest is usually impaired and raised in pitch, and a wheezing, whistling sound is, in some cases, heard over the position of the compressed tube. Pressure on the main bronchus of the left side, is accompanied by corresponding alterations in the left lung.

Pressure on the *root of the lung*, more especially, it is supposed, on the *branches of the pulmonary plexus of nerves*, is occasionally followed by destructive inflammation of the lung tissue. In some of these cases the lung is found collapsed after death, and studded with small abscesses. In a case,

which has quite recently come under my observation in the *post-mortem* theatre, the lower lobe of the left lung was consolidated and riddled with cavities, the result of the pressure of the sac of a large aneurism; hæmoptysis, which ultimately proved fatal, was caused by the rupture of the sac into one of these cavities; the upper lobe of the left lung, and the right lung were, with the exception of emphysema, perfectly healthy. In cases of this description, the physical signs of collapse, or breaking down of the lung, may sometimes be observed during life. Pressure on the *trachea* is, in many cases, attended, as has been previously remarked, by stridor.

On auscultating the trachea, a blowing murmur, synchronous with the action of the heart, can be heard in some cases of aneurism, as Dr D. Drummond has pointed out. It seems to be an exaggeration of the shock, which is usually communicated to the air in the chest by the expansion and contraction of the heart.

Pressure on the left recurrent laryngeal nerve often produces paralysis or spasm of the left vocal cord, conditions which can, of course, be readily recognised by the laryngoscope.

Pressure on the œsophagus produces dysphagia, which may be demonstrated by making the patient swallow a piece of solid food, and by observing the result, and by auscultating the œsophagus during the act of swallowing solid and liquid substances. An œsophageal bougie must not be passed in cases of this description.

Pressure upon the fibres of the sympathetic, more especially those fibres which are connected with the lower cervical and first dorsal ganglia, produces alterations in the size of the pupil and in the vaso-motor condition of the head and neck. These alterations are usually restricted to one side, since, as a rule, the nerves on one side only are implicated. The exact nature of the pupil and vaso-motor modifications depends upon the manner in which the sympathetic fibres are affected. *Irritation* of the cilio-spinal branches of the sympathetic on one side produces—(a) spasm of the dilator muscle of the pupil (which is supplied by the sympathetic), and (b) contraction of the blood vessels of the corresponding side of the head and neck, manifested externally by pallor and dryness of the skin

and diminution of temperature. *Destruction* of the cilio-spinal branches of the sympathetic, causes (a) paralysis of the dilator muscle of the iris and contraction of the pupil (in consequence of unopposed action of the pupil sphincter, which is supplied by the third nerve), and (b) dilatation of the blood vessels of the head and neck on the affected side, conditions which are manifested externally by congestion, elevation of temperature, and unilateral sweating.¹ Too much importance must not, however, be attached to differences in the size of the pupil, for inequality in the size of the pupils is of frequent occurrence even in health, and may be due to many different pathological conditions. When combined with an altered vaso-motor condition of the side of the head and neck, inequality in the size of the pupils is only suggestive of intra-thoracic pressure; and even when it is proved to be the result of intra-thoracic pressure, it does not necessarily follow that an aneurism is the cause of that pressure, for the pressure may be due to a solid intra-thoracic growth.

Downward displacement of the liver is observed in some cases in which the thoracic aneurism is of large size.

Summary of the Chief Symptoms and Physical Signs which are met with in Aneurisms of the different parts of the Thoracic Aorta.

In order to complete this description, it may perhaps be well to sum up the *chief* symptoms and physical signs, which result from aneurisms of the root of the aorta, and of the ascending, transverse, and descending portions of the vessel respectively.

¹ 'This dilating influence of the sympathetic may, as in the case of the vaso-motor action of the same nerve, be traced back down the neck, along the rami communicantes and roots of the last cervical and first dorsal or two first dorsal spinal nerves, to a region in the lower cervical and upper dorsal cord (called by Budge the centrum cilio-spinale inferius), and from thence up through the medulla oblongata to a centre, which, according to Henson and Völckers, lies in the floor of the front part of the aqueduct of Sylvius.'—(*A Text-Book of Physiology*, by Professor Michael Foster, p. 466, third edition.) Professor Ferrier tells me that his observations show that the dilating fibres pass out of the spinal cord through the anterior root of the second, and perhaps in some cases of the first, dorsal nerves. I have not been able to find the paper to which Professor Ferrier referred me, in the *Philosophical Transactions*.

1. *Aneurism of the root of the aorta involving the sinuses of Valsalva.*—In cases of this description, the aneurism is often entirely latent, *i.e.* it may give rise to no symptoms, and may not be attended with any distinct signs. Incompetence of the aortic valves is often present. In some cases, attacks of angina pectoris (due to irritation of the branches of the cardiac plexus) are observed. Pericarditis is frequently produced by the irritative pressure of the sac, and is a highly satisfactory result, for, in consequence of the adhesions, the aneurism is unable to rupture into the sac of the pericardium—a common termination of aneurisms in this situation, in those cases in which the pericardium is unadherent. In rare and exceptional cases the aneurism may burst into the pulmonary artery, or into the left auricle of the heart (see fig. 281). Aneurisms of the sinuses of Valsalva rarely attain to any size; the case represented in figures 282, 283, 284, 285, and 286, is, so far as I know, quite unique. Should the aneurism spring from the anterior surface of the aorta, it may compress the pulmonary artery, and possibly give rise to symptoms and physical signs of pulmonary constriction.

2. *Aneurisms of the ascending portion of aortic arch, above the sinuses of Valsalva.*—This is the part of the thoracic aorta which is most frequently affected—the ‘*seat of election*’ of aortic aneurisms, as I am in the habit of terming it. The aneurism usually makes its way forwards, and soon comes in contact with the front wall of the chest; it is often of large size, and is, as a rule, very readily detected on physical examination. The chief pressure signs and symptoms to which it gives rise, are (*a*) pain; (*b*) dyspnœa, cough, and alterations in the right lung from pressure on the pulmonary tissue or right bronchus; and in some cases (*c*) engorgement of the veins of the head and neck from pressure on the superior vena cava. Aneurisms in this situation often erode the front wall of the chest.

3. *Aneurisms of the transverse portion of the aortic arch* are very common, though not so common as aneurisms of the ascending portion. The symptoms and signs are, as a rule, well marked and characteristic, and the diagnosis easy. The tumour sometimes makes its way up into the neck

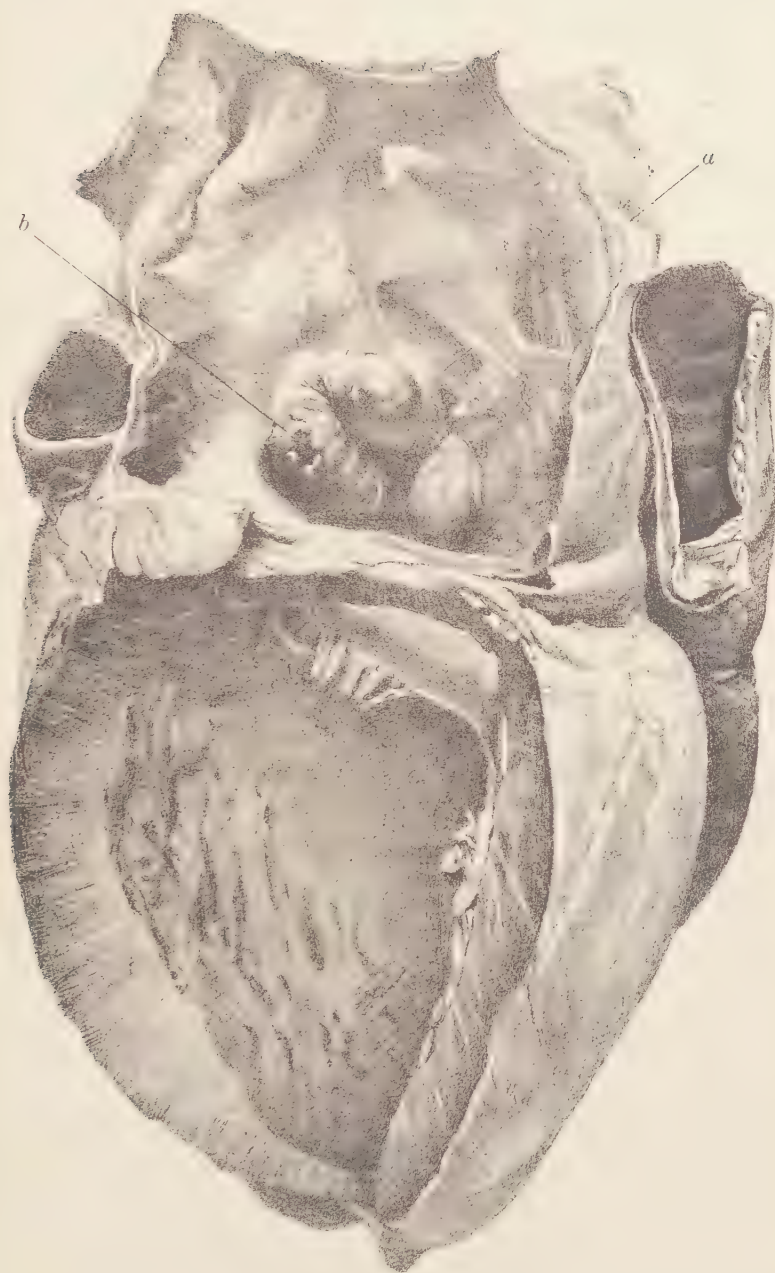


FIG. 281.—*Aneurism of the commencement of the aorta rupturing into the left auricle. (Natural size.)*

The left auricle and left ventricle have been cut open from behind, a bridge of muscular tissue (*b*) being left between them.

The letter *a* points to three projections which the aneurism has formed in the anterior wall of the auricle; *b*, to a communication between the sac of the aneurism and the cavity of the auricle.

(The specimen is in the Anatomical Museum of the Edinburgh University, and is represented by Professor Turner's permission.)



FIG. 282. *Portrait of J. S., showing the position and size of the External Tumour when the patient first came under observation. (Seen from the right side.)*



FIG. 283. *Portrait of J. S., showing the position and size of the External Tumour when the patient first came under observation. (Front view.)*

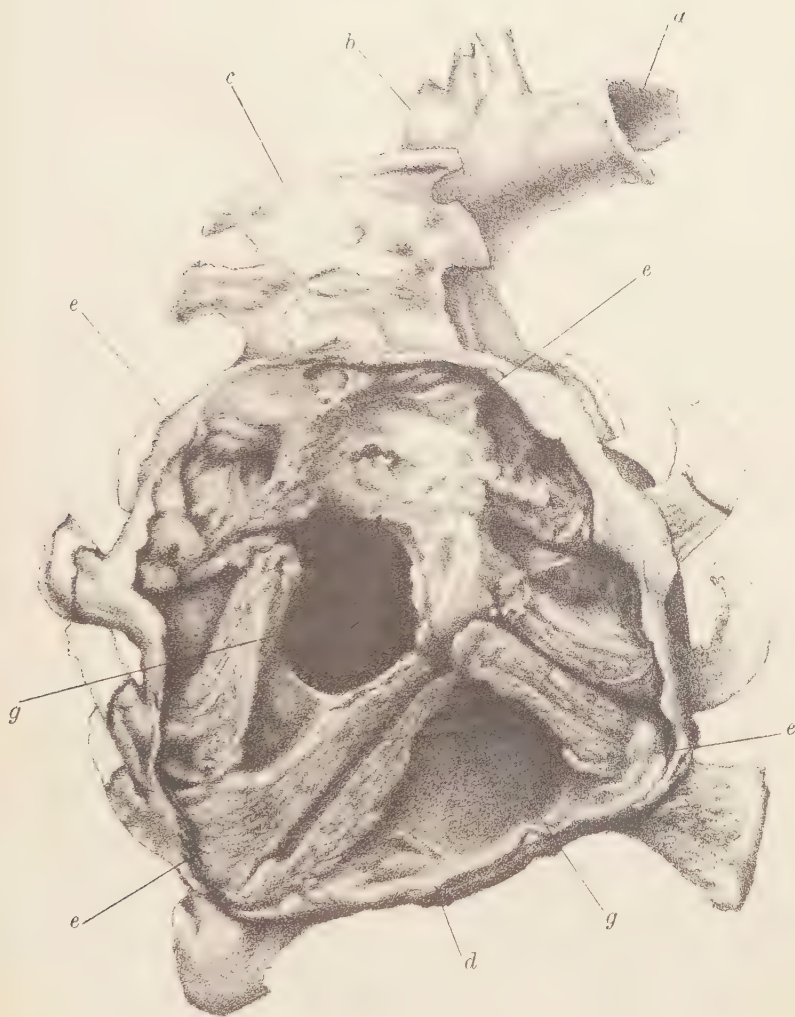


FIG. 284. *Sternum and adjacent ribs, showing the apertures of perforation, and the outline of the base of the false Aneurism formed externally in the case of J. S.*

The letter *a* points to the termination of the transverse portion of the aortic arch; *b*, to the innominate artery; *c*, to the manubrium sterni; *d*, to the ensiform cartilage; *e, e, e, e*, to the outline of the base of the false aneurism; *g, g*, to the eroded apertures in the chest wall.

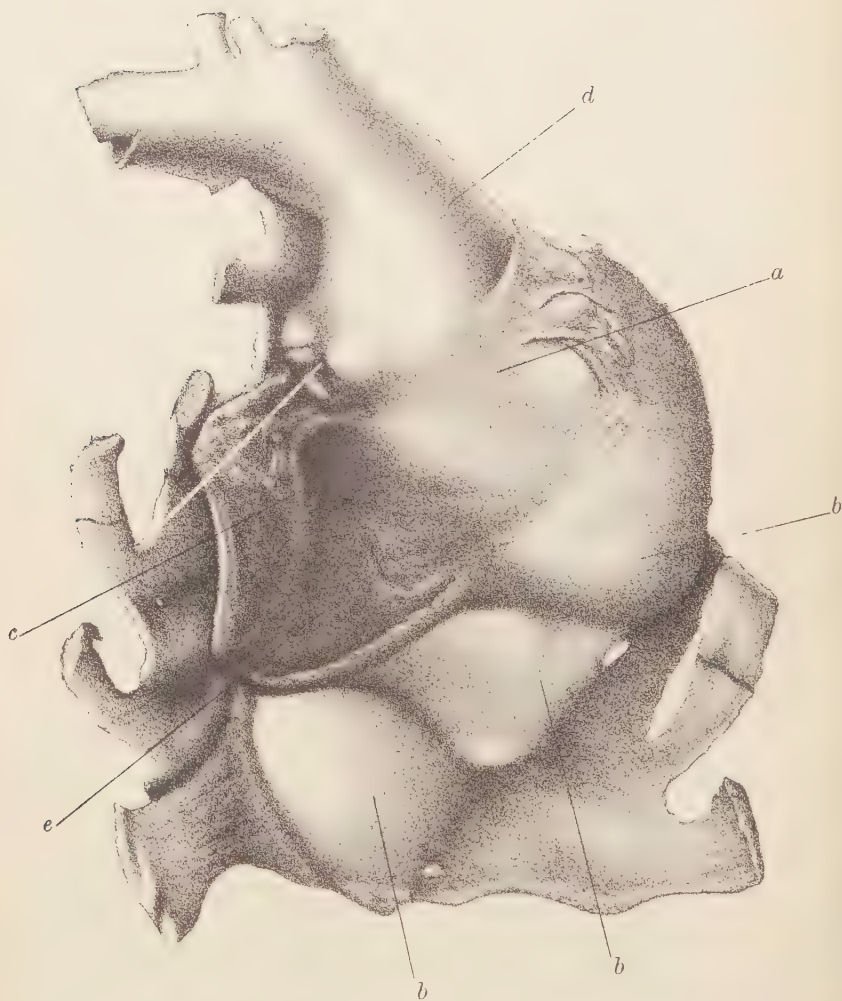


FIG. 285. *Aneurism springing from the very commencement of the Aortic Arch, and causing erosion of Chest Wall and death by external rupture. Seen from behind. (Case of J. S.)*

A probe has been passed into the aortic orifice; the left ventricle has been cut away, and the interior of the right ventricle exposed. The sac of the aneurism is seen to be in close contact with the exterior of the right auricle, right ventricle, and pulmonary artery.

The letter *a* points to the aneurism at its point of origin at the root of the aorta; *b, b, b*, to the secondary tumours which it has formed, and which are in contact with the chest wall; *c*, to the orifice of the pulmonary artery; *d*, to the aortic arch above the aneurismal sac; *e*, to the interior of the right ventricle.

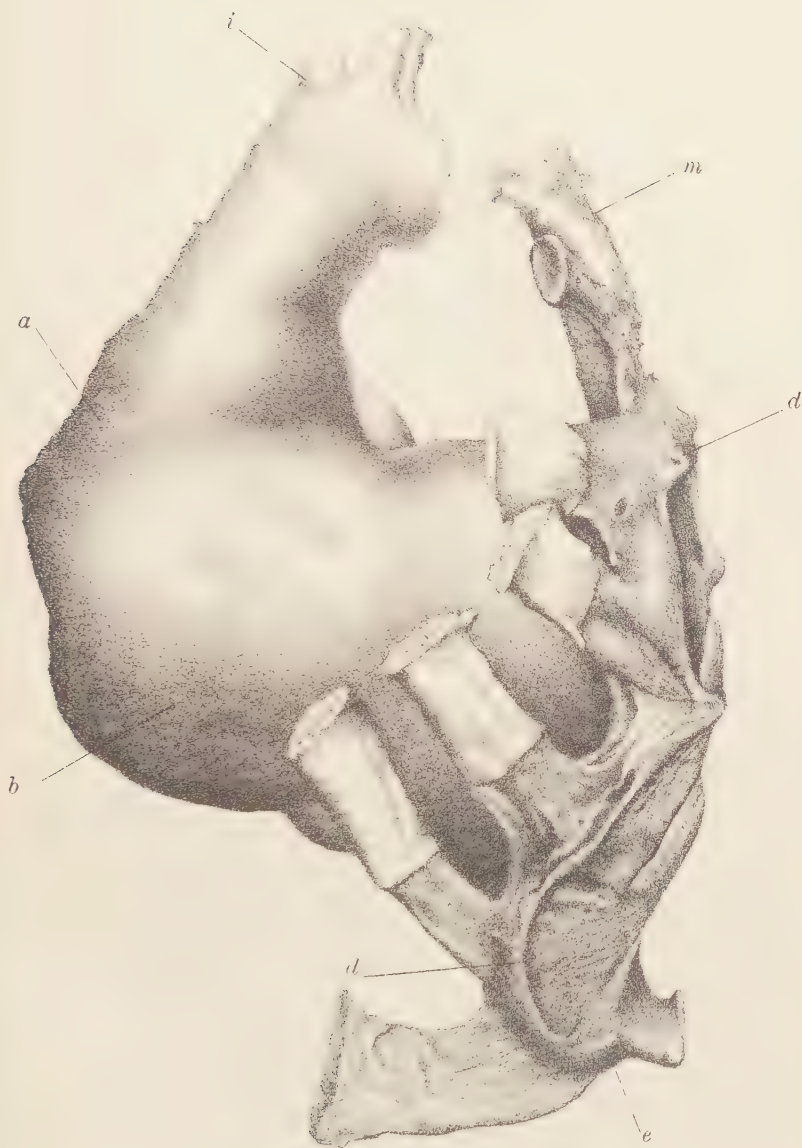


FIG. 286. Aneurism springing from the commencement of the Aortic Arch, and causing erosion of the Chest Wall in the Lower Sternal Region, and eventually producing the large external tumour represented in figs. 282 and 283. (The aneurism and the bony wall of the Thorax, as seen from the right side.)

The letter *a* points to the ascending portion of the arch of the aorta, from the commencement of which the aneurism *b* springs; *i*, the innominate artery; *m*, the manubrium sterni; *d*, *d*, the base of the external tumour; *e*, the ensiform cartilage.

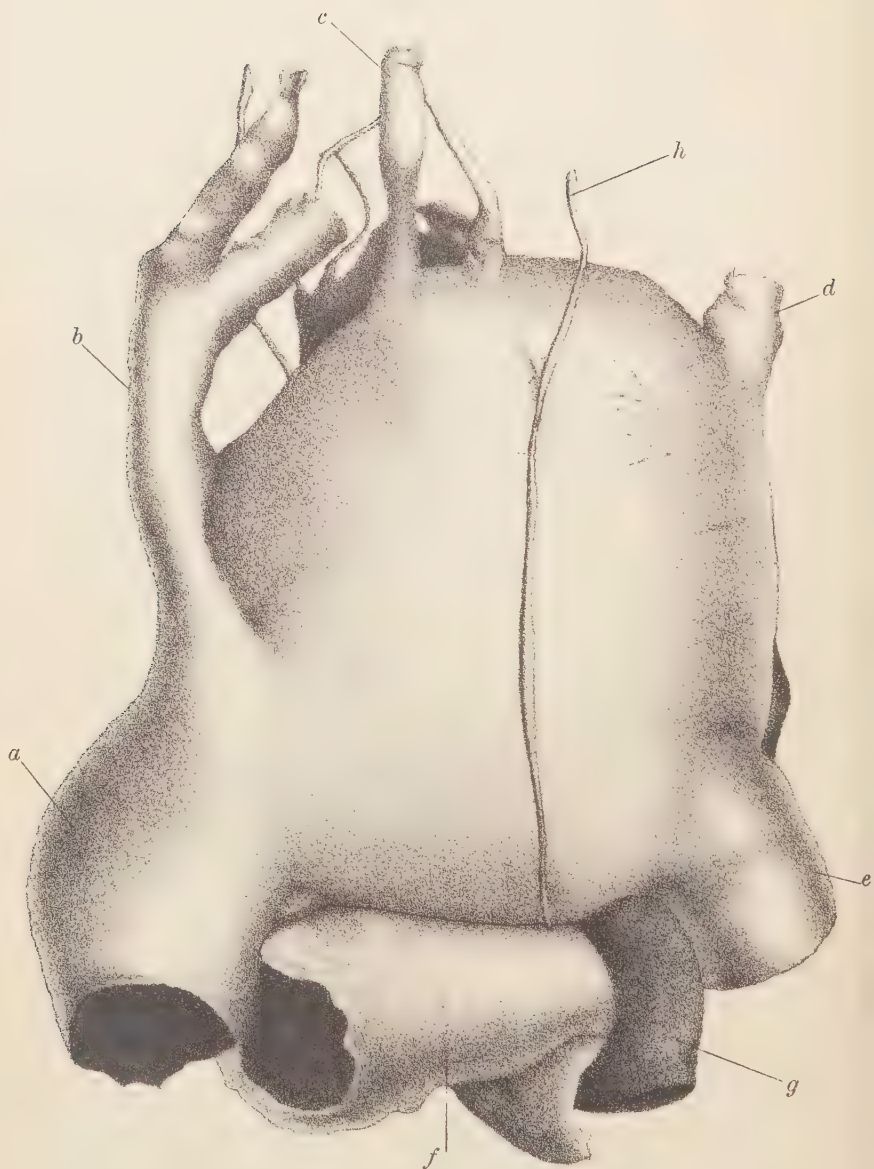


FIG. 287. *Aneurism of the transverse portion of the Aortic Arch, which produced great difficulty in breathing from pressure upon the Trachea and Left Recurrent Laryngeal nerve. Death resulted from rupture into the Trachea. (Front view, natural size.)*

The letter *a*, points to the ascending portion of the aortic arch; *b*, to the innominate artery; *c*, to the left common carotid artery; *d*, to the left subclavian; *e*, to the descending portion of the aortic arch; *f*, to the pulmonary artery; *g*, to the œsophagus; *h*, to the left pneumogastric nerve.

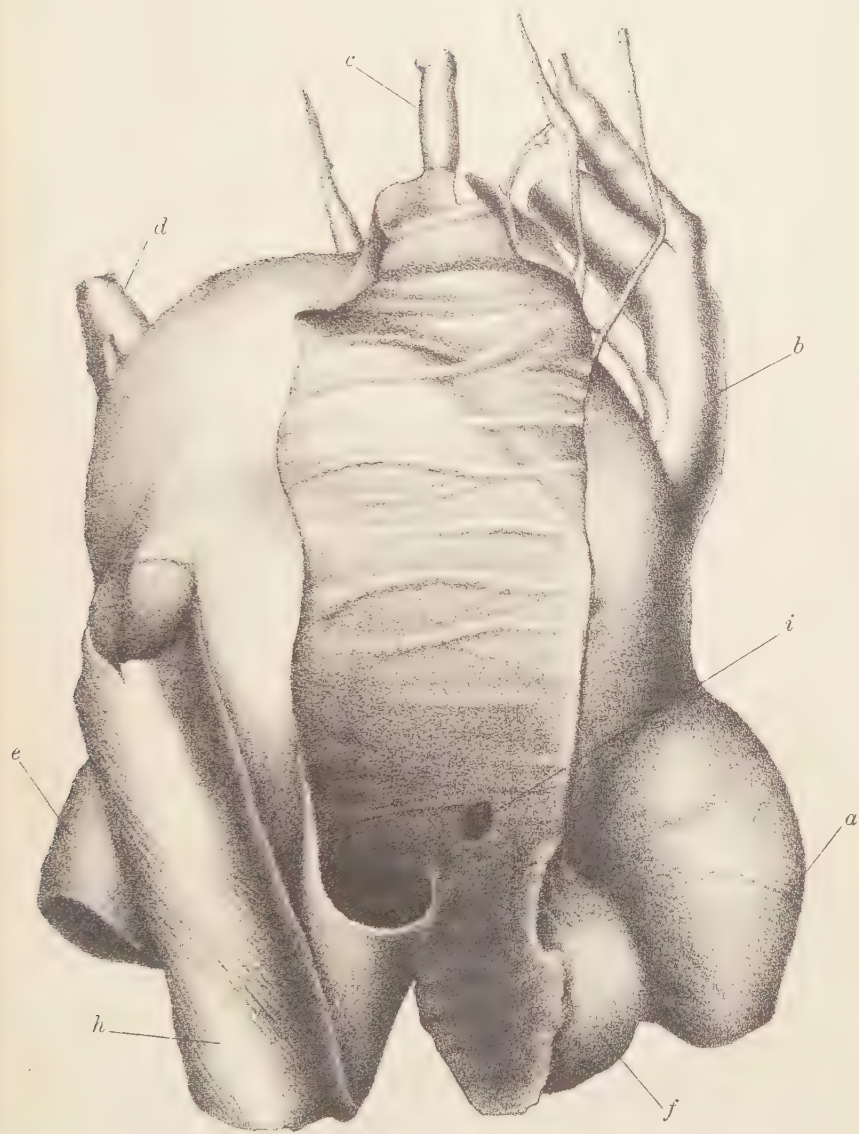


FIG. 288. *Aneurism of the transverse portion of the Aortic Arch, which produced great difficulty in breathing from pressure upon the Trachea and left Recurrent Laryngeal Nerve. Death resulted from rupture into the Trachea. (Back view, natural size.)*

The letter *a* points to the ascending portion of the aortic arch; *b*, to the innominate artery; *c*, to the left common carotid artery; *d*, to the left subclavian; *e*, to the descending portion of the aortic arch; *f*, to the pulmonary artery; *h*, to the oesophagus; *i*, to the aperture of rupture in the trachea, which is situated immediately above the bifurcation of the bronchi.

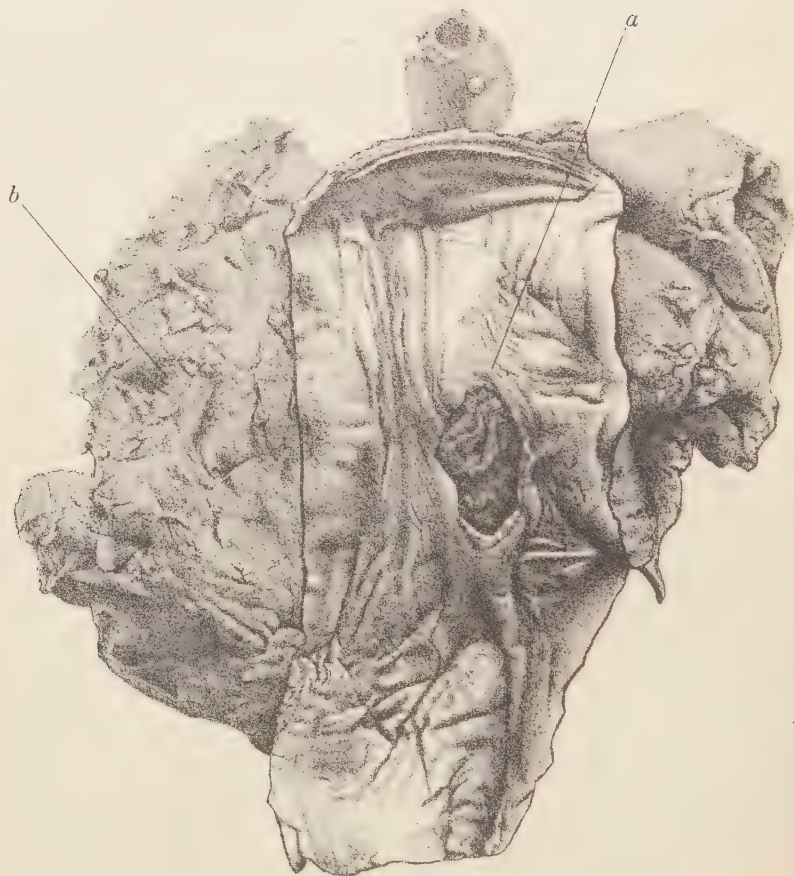


FIG. 289.—*Aneurism of the thoracic aorta, rupturing into the œsophagus. (Natural size, seen from behind.)*

The letter *a*, points to the œsophagus just above the orifice; *b*, to the aneurism. The sac was almost completely filled with laminated fibrine, the clot can be seen through the orifice in the œsophagus.



FIG. 290.—*Erosion of the spinal column due to the pressure of an aneurism. (Natural size.)*

The anterior surface of the bodies of two vertebrae are extensively destroyed, and the spinal canal is exposed.



FIG. 292.

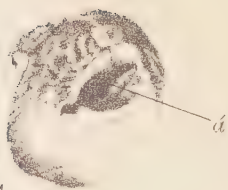


FIG. 294.

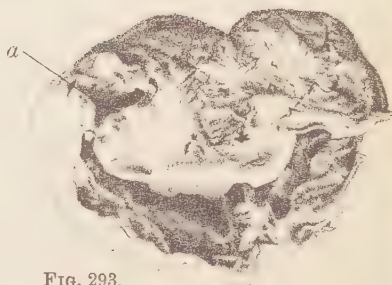


FIG. 293.



FIG. 291.

FIG. 291. Clot from the interior of a large aneurism of the *Innominate* artery. (Natural size.)

FIG. 292.—Laminated blood clot from an aneurism of the *popliteal* artery in a boy who suffered from cardiac disease. (Natural size.)

The aneurism had undergone a spontaneous cure; the lamination of the clot is well shown.

FIGS. 293 and 294.—Clots from aneurisms of the *innominate* and *left common carotid* arteries. (Natural size).

A narrow channel, *a*, *a'*, passes through the centre of each clot.

(see fig. 287); its pulsation can usually be felt in the supra-sternal notch; alterations in the characters of the two radial or the two carotid pulses are frequently observed, for one or other of the large branches springing from the arch is often implicated in the sac or obstructed. The œsophagus, trachea, recurrent laryngeal nerve, and the left innominate vein are the parts which are most liable to be pressed upon. The aneurism often proves fatal by rupturing into the trachea or œsophagus. (See figs. 288 and 289.)

4. *Aneurisms of the descending portion of the thoracic arch and of the descending thoracic aorta* are often latent, and until they have perforated the chest wall (which they sometimes do) they are very difficult to detect by means of physical examination. A dull, boring, localised pain in the back or spinal column is very generally present, and is highly characteristic. The œsophagus and root of the left lung are very likely to be pressed upon. The thoracic duct and azygos vein are often also implicated. A systolic murmur over the seat of the pain in the back is highly suggestive. Aneurisms of this part of the aorta often rupture into the left pleura, or into the œsophagus (see fig. 289); they very frequently erode the bodies of the vertebræ (see fig. 290¹), and may finally press upon the spinal cord, or burst into the spinal canal. In some cases they perforate the chest wall, usually to the left side of the spinal column.

Course and terminations.—The average duration of aneurisms of the thoracic aorta is said to be about two years from the time when the condition is first recognised; but the course and duration are very uncertain, for, on the one hand, the condition may at any moment prove fatal, and on the other, cases have been met with, in which the subjects of thoracic aneurism have for years been able to follow their ordinary occupations without any very great inconvenience.² The termination is

¹ In this case the aneurism involved the abdominal aorta.

² Balfour relates a case in which the patient maintained himself for ten years as a hotel porter, with a large aneurism projecting through the walls of the chest. —*Diseases of the Heart*, p. 405.

almost invariably fatal. In extremely rare cases, the aneurism consolidates and is cured. Death is generally caused by rupture of the sac. The aneurism may burst into the pleura, lung tissue, trachea, bronchi, œsophagus, pericardium, through the diaphragm, or externally. In a minority of cases death is due to gradual and progressive exhaustion and inanition, or to some inter-current complication.

Diagnosis.—The steps in the diagnosis of a supposed case of aneurism of the thoracic aorta are:—

1. Is a thoracic aneurism present?
2. If a thoracic aneurism is present, is it an aneurism of the thoracic aorta?

Step No. 1. Is a thoracic aneurism present?

This question is in some cases decided with the greatest ease, in others, the diagnosis is difficult or impossible. When the aneurism is small and deep seated, and when there are no pressure symptoms, the diagnosis is impossible. Many aneurisms of the sinuses of Valsalva come under this head. In cases of this description, the aneurism may burst into the pericardium and suddenly cause the death of a patient who had previously made no complaints, and who had presented up to the time of death all the appearance of robust health. The case from which the specimen represented in fig. 270 was drawn, is an excellent illustration in point. When, on the other hand, the aneurism has perforated the chest wall, and formed an external pulsating tumour, the diagnosis is self-evident. When the aneurism is large, and more especially when it is in contact with the chest wall (even although it has not perforated), the diagnosis is generally easy.

The physical signs which are of greatest importance from a diagnostic point of view are—(1) a circumscribed dull area in the course of the aorta; (2) pulsation over the dull area, more especially pulsation of an expansile character, synchronous with the cardiac systole, and at least as forcible as the pulsation of the heart; (3) well marked auscultatory phenomena, more particularly a systolic murmur with an

accentuated second sound, or an accentuated second sound with a normal, feeble or toneless first sound, over the dull area; and (4) pain and other pressure symptoms. When these symptoms and signs are met with *in combination*, the diagnosis does not present any difficulty. Difficulties in diagnosis chiefly arise in those cases in which some only of the physical signs just mentioned are present.

The conditions which are most likely to be confounded with aneurisms of the thoracic aorta may be conveniently divided into two groups, viz. :—

Group 1. Conditions in which some of the physical signs of thoracic aneurisms (more especially localised dulness on percussion and pulsation in the course of the aorta) are present, but in which there are no pressure signs.

Group 2. Cases in which there are pressure signs, together with some of the physical signs of thoracic aneurism.

The chief conditions included under these groups (which run into each other) are :—

1. *Simple dynamic pulsation of the thoracic aorta.*—This is certainly a rare condition—infinately more rare than simple dynamic pulsation of the abdominal aorta, which is not unfrequently mistaken for an aneurism of the abdominal aorta—but it does occasionally occur. It was probably the cause of very distinct pulsation, and some dulness in the second right interspace, in a case which I saw recently in consultation with Dr William Murray of Newcastle-on-Tyne.

The patient, a very robust and healthy-looking young man, came under Dr Murray's care, complaining of palpitation, and a feeling of uneasiness in the region of the heart; pain and pressure symptoms were entirely absent, but pulsation and dulness over the aorta were so distinct as to lead Dr Murray—whose diagnostic ability generally, and in aneurism in particular, is well known—to believe that an aneurism of the ascending portion of the aortic arch was probably present. After some months of treatment, the local indications of aneurism gradually subsided, and when I saw the case they had completely disappeared. The pulse throughout the

course of the attack, and at the time of my visit, was unduly slow, but there were no physical signs of organic cardiac disease. So far as Dr Murray and I could judge, the condition was probably neurotic. In these cases the absence of pain and other pressure signs is a point of cardinal importance. The fact that there is no evidence of organic disease of the heart and arteries; the age and sex of the patient (neurotic pulsation being almost exclusively confined to young adults, and being much more common in women than in men); and the presence of symptoms and signs indicative of deranged cardiac innervation, such, for example, as an uneasy feeling in the region of the heart, consciousness of the heart's action, and palpitation, are the points on which reliance must be placed in making the diagnosis.

2. *Displacement of the aorta the result of spinal curvature.*—

Curvature of the spine not unfrequently displaces the aorta forwards and to the right. Some pulsation and dulness may be present in the second right interspace, and may lead to the supposition of aortic aneurism. When the patient is in good health, more especially when the heart and arteries are healthy, the diagnosis does not present much difficulty. Great importance is to be attached to the absence of pain and other symptoms of intra-thoracic pressure. In those cases in which the heart is diseased, the diagnosis may be most difficult, and so far as my experience enables me to form an opinion, disease of the heart and of the aorta is of frequent occurrence in those cases in which the upper outlet of the thorax is narrower than normal.

The following case, which I reported some years ago in the *Lancet* (March 9th, 1878, page 346), illustrates the difficulties in diagnosis in some cases of this description.

Case.—*Malposition of the Aorta due to Rickets, simulating Aneurism of the Arch of the Aorta.*

W. W., æt. 46, an engineer, was admitted to the Newcastle-on-Tyne Infirmary under my care, on June 10th, 1875, suffering from orthopnœa and general dropsy.

Diagnosis of Aneurism of the Thoracic Aorta. 725

Previous history.—He enjoyed good health until he was thirty-five years of age. He was then laid up with bronchitis and pleurisy. The bronchitis has returned every now and again since. The present attack commenced three months ago with cough and expectoration. He has been confined to bed for seven weeks. Dropsy set in six weeks ago. His spine has been very much curved since he was a little boy.

Condition on admission.—There is great general dropsy. He is very short of breath. The face and upper extremities are markedly cyanotic. The external jugular veins are distended. The root of the neck looks swollen, and there seems to be a constriction of the upper part of the thorax. The chest is prominent in front, more so on the left than on the right side. The spine in the lower cervical and upper dorsal regions is very much curved, the direction of the curvature being forwards and to the right. The præcordia is prominent. Dull heaving pulsation is felt all over the præcordial area, and in the second and third intercostal spaces both to the right and left of the sternum. There is strong pulsation in the supra-sternal notch. The pulsation is most marked in the second left intercostal space just outside the sternum. There is marked dulness on percussion over the manubrium, and on both sides of it over the area of pulsation described above. The area of cardiac dulness is considerably increased, measuring four and a half inches transversely. On auscultation over the manubrium and area of pulsation to the right and left of it, the cardiac sounds are heard, valvular and superficial in character, the second being decidedly accentuated. These characters are most marked over the area of maximum pulsation in the second left intercostal space. At the cardiac apex the sounds are normal. The radial pulse numbers 120; it is regular, very much weaker in the left than in the right wrist. Respiration in the left lung is weaker than in the right. Bronchitic râles are heard all over the chest, and there is some dulness over the bases of the lungs (hydrothorax). With the exception of the venous obstruction already described, there are no pressure symptoms.

It is unnecessary to give the daily progress of the case; suffice it to say, that the patient gradually got worse, and died on June 16th.

Autopsy twenty-one hours after death.—The anterior curvature of the spine was very great, the distance between the second right sterno-costal articulation and the spinal column being only one inch and a half. All the structures passing in and out of the superior outlet of the thorax were compressed. The aorta was displaced forwards and towards the left. The œsophagus passed tolerably freely along the left side of the projecting vertebræ. The pericardium was universally adherent, the adhesions being recent. The heart weighed fourteen ounces and a half. The aortic valve was slightly incompetent, and was covered with minute vegetations. The aortic arch was normal. The lungs presented the ordinary appearances seen in acute bronchitis. There was a considerable

amount of serous fluid in each pleural cavity. There was no apparent cause for the inequality of the pulses, nor for the inequality of the respiratory murmur on the two sides.

3. *Pulsating empyema*.—This condition can usually be distinguished without difficulty after a careful examination of the case. The pulsation is less forcible than the pulsation of the heart; there are no pressure symptoms; the dulness on percussion and the pulsation may not lie in the course of the aorta, but even when they are so situated, the absence of other indications of cardiac or arterial disease on the one hand, and the presence of constitutional derangement and of local evidence of disease of the lung or pleura, on the other, enable us usually, without much difficulty, to come to a correct conclusion as to the nature of the case.

4. *Solid intra-thoracic tumours*.—In some of these cases, the tumour is in contact with the chest wall on the one hand, and with the aorta on the other; in others the tumour is deeply situated. In the latter case, more especially, the differential diagnosis is often extremely difficult or impossible.

When the tumour is in contact with the chest wall on the one hand and the aorta on the other, localised dulness over the course of the aorta is present; there is often an impulse communicated from the aorta over the seat of the dulness; a systolic murmur, due to constriction of the aorta by the pressure of the tumour is frequently present, and symptoms and signs of intra-thoracic pressure may be prominent. Obviously, therefore, an intra-thoracic aneurism will, in many cases, be closely simulated. A careful observer can, however, usually come to a correct conclusion as to the nature of the case. The dulness due to a solid tumour is seldom so localised as it usually is in thoracic aneurisms; the impulse is usually slight, and never, so far as I know, equals the force of the cardiac impulse (as the impulse of a thoracic aneurism often does); the chest wall is rarely if ever eroded by a solid growth arising within the thorax; the systolic murmur due to the pressure of a solid intra-thoracic tumour on the aorta is not usually accompanied by an accentuated second sound, in fact the aortic second sound is generally faint or absent at the

seat of the murmur; the breath sounds—often in the form of bronchial or tubular breathing—can generally be heard over the dulness due to a tumour, but are absent over the dulness due to an aneurism; though pressure symptoms are prominent, pain is seldom great in cases of solid intra-thoracic growth, indeed, in many cases there is no pain throughout the course of the case; constitutional symptoms are generally much more marked in solid growths. But these and other points will be presently considered in detail.

In a case, which I have recorded in the *British Medical Journal*, vol. i., p. 8, 1877, the signs of thoracic aneurism were very closely simulated, but space does not allow me to give the details.

Cystic tumours are occasionally, though very rarely, met with in the cavity of the thorax. In the following case the cyst was in contact with the arch of the aorta, and the physical signs of aortic aneurism were closely simulated. The reasons which led me ultimately to exclude a true sacculated aneurism are set forth in the remarks attached to the record of the case.

Case of Cystic Tumour in the Anterior Mediastinum simulating Aneurism.

A. C. æt. 50, single, a drayman, formerly a soldier, was first admitted to the Newcastle-on-Tyne Infirmary, under my care, on 17th February 1876, suffering from acute albuminuria of three weeks' duration.

Twenty years previously he had suffered from syphilis. For several years past he had been a hard drinker.

On examining the thorax, visible and tangible pulsation was perceived in the second right interspace. A slight systolic thrill could be felt when the hand was placed over the same spot, and there was well-marked and limited percussion dulness. A systolic murmur and somewhat accentuated second sound were heard on auscultation. (The thrill, systolic murmur, and accentuation of the second sound were perhaps better marked over the mid-sternum at the level of the fourth costal cartilage than over the area of pulsation.)

There were no pressure signs; it was particularly noted that there never had been any pain. Shortness of breath and cough were complained of, but were evidently due to the presence of bronchial catarrh.

The heart was of normal size, the apex being situated an inch immediately below the left nipple.

The diagnosis, as regards the thoracic lesion, was an aneurism of the aortic arch.

On 6th April the patient discharged himself. The urine still contained a trace of albumen, the physical signs at the seat of the supposed aneurism being the same.

On 5th October he was again admitted, suffering from a relapse of the renal dropsy; and after remaining in hospital for a month was again discharged. The thoracic physical signs were still unchanged.

He continued well until September 1877, when he began to suffer from pain in the region of the stomach and from difficulty in swallowing.

On 14th November 1877 he was re-admitted under my care. He was now greatly emaciated, his expression was haggard and anxious, and he presented a remarkably cachectic appearance.

The physical signs within the thorax were as before, except that the apex-beat was somewhat elevated, corresponding to the left nipple.

Pain and tenderness on pressure were complained of in the epigastric region, but no tumour could be perceived. The epigastric pain was increased immediately after taking food. There had been no vomiting. The stomach seemed of normal size. There was marked dysphagia, the stoppage being referred to a point corresponding to the lower end of the sternum.

The right pupil was only half the size of the left. He complained of numbness in the fingers of the right hand.

The urine was copious, pale, sp. gr. 1010. It contained $\frac{1}{8}$ albumen, and a few granular and hyaline casts.

I now discarded the notion of a true sacculated aneurism in favour of a general dilatation of the aortic arch. This opinion was based upon the continued absence of all pressure signs—especially pain—and the unchanged condition of the physical signs at the seat of pulsation.

On 21st January the patient was seized with a severe epileptiform convulsion, and died twelve hours afterwards.

The autopsy was made sixteen hours after death. The body was much emaciated. A cystic tumour, the size of a hen's egg, was situated in the anterior mediastinum, in immediate contact with the ascending, and the junction of the ascending and transverse portions of the aortic arch. (The specimen was exhibited to the Edinburgh Medico-Chirurgical Society, May 1878.) The anterior surface of the tumour was partly covered by lung tissue, the uncovered portion being in contact with the chest-wall at a point corresponding to the second right interspace. The cyst contained a clear, watery-looking fluid of neutral reaction, and of sp. gr. 1010. The fluid was almost entirely coagulated by heat and nitric acid. On standing, it deposited a scanty sediment containing a few leucocytes, but no other formed elements.

The aorta was somewhat dilated and atheromatous at its base. The aortic valves were thickened and cartilaginous, but competent.

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The heart weighed 13 oz.

The stomach was filled with a huge clot of black blood. A large ragged malignant ulcer surrounded the œsophageal opening. The coats of the stomach were very much thickened at the seat of the ulcer. The orifice of the œsophagus was partly obstructed by the new growth. The kidneys were in an early stage of the large white form of Bright's disease.

Remarks.—In the works at my disposal I cannot find any reference to the occurrence of a simple serous cyst in the anterior mediastinum. The case is obviously, therefore, of great pathological rarity. It is, however, in its clinical aspects that it is chiefly interesting; indeed, I have not, either in reading or in practice, come across any case in which the direct physical signs of an aortic aneurism were so closely simulated. The pulsation and limited dulness at the 'seat of election' of aortic aneurisms resulted, of course, from the presence of the tumour and from the way in which it was related to the aortic arch. The accentuated second sound was due, chiefly, I think, to the increased arterial tension which resulted from the kidney disease, partly to the dilated condition of the aortic arch. The systolic murmur and thrill were evidently due to the condition of the aorta.

In those cases in which the solid intra-thoracic tumour or aneurism is deeply situated, the physical signs are of course much more indistinct; in many cases the diagnosis is extremely difficult, and can only be arrived at by a judicial survey of *all* the facts and probabilities of the case, viz. :—

- (1.) The relative frequency of the two diseases.
- (2.) Certain general considerations, such as the age, sex, and occupation of the patient.
- (3.) His hereditary tendencies.
- (4.) His previous history.
- (5.) His present condition (general appearance, symptoms, physical signs, associated diseased conditions).
- (6.) The progress of the case.
- (7.) The influence of treatment.

I will now consider each of these points in detail. My remarks are based upon a comparison of the cases of aneurism analysed by the late Dr Sibson, and recorded in his great work on Medical Anatomy; of the cases of aneurism tabulated by the late Dr Hayden; and of all the cases of primary intra-thoracic cancer and lympho-sarcoma which I could find recorded in the *Lancet*, *British Medical Journal*, *Medical*

Times and Gazette, and *Transactions of the Pathological Society of London*, during a period of fifteen years. (This analysis was made several years ago, and as a few of the journals were missing from the library of the Newcastle-on-Tyne Infirmary when I made the search, and as it is not therefore strictly accurate or brought up to date, I omit the figures; but the main results may, I think, be relied upon.)

Relative frequency of aneurism and tumour.—Aneurism is much more frequent than tumour. (My own experience confirms this statement very strongly, but it must be remembered that in the neighbourhood of Newcastle, in which my experience has been chiefly gained, aneurisms abound.)

Age.—Aortic aneurisms are most frequent between the ages of thirty and fifty, and are extremely rare before twenty. Intra-thoracic tumours (I have limited the inquiry, it must be remembered, to cancers and lympho-sarcomatous growths) may occur at any age, but are not uncommon before the age of twenty; they occur most frequently between the ages of twenty and fifty, and seem to be met with almost in equal numbers between the three periods of ten years—from twenty to thirty, from thirty to forty, and from forty to fifty.

Sex.—Aneurisms are eight times more frequent in males than in females. Tumours occur almost as frequently in females as in males.

Occupation.—Aneurism is much more common amongst those whose occupations necessitate hard manual labour, and in soldiers, sailors, and prostitutes. Tumour seems also more common in the lower orders, but it is not more frequent in soldiers, sailors, prostitutes, and persons who follow laborious occupations, than in other people.

Hereditary tendencies and temperament.—Aneurisms occur most frequently in persons of a sanguine temperament, and in those families in which diseases of the heart and vascular system are hereditary. Lympho-sarcomata affect persons of a lymphatic and scrofulous habit. A hereditary history of cancer cases can often be obtained in persons suffering from intra-thoracic cancer.

Ætiology.—Strain, syphilis, alcoholic excesses, rheumatism,

and all conditions which produce arterial degeneration or increased arterial tension, tend to produce aneurism. The causes of tumour are often obscure. In some cases of intra-thoracic cancer, direct injury seems to have been the exciting cause. Lympho-sarcomata seem sometimes to be due to local irritation. In one of my own cases the only obvious cause was the inhalation of gunpowder smoke. In other cases the tumour is secondary to a primary cancer or sarcoma in some other part of the body. The intra-thoracic tumours which are likely to be mistaken for aortic aneurisms are, in the majority of cases, primary, and originate in the bronchial glands or the remains of the thymus. Secondary intra-thoracic tumours usually occur as nodules (cancerous infarctions in many cases) in the pulmonary tissue. In cases of this description the symptoms and signs of thoracic aneurism are rarely simulated.

History and mode of development.—In a large proportion, probably in at least 50 per cent. of the cases of aneurism, there is a history of syphilis. In cases of tumour a history of syphilis is not more frequent than in the general average of patients. In aneurism the symptoms may develop suddenly. In tumour the symptoms almost always develop slowly and gradually.

General appearance.—Patients suffering from aneurism often present all the external appearances of perfect health. The subjects of intra-thoracic cancer or lympho-sarcoma are usually pale, emaciated, and cachectic; it is quite exceptional to find them presenting the appearances of health. (In some cases of aneurism the expression is anxious, the patient looks ill, and may be emaciated. These symptoms are seldom, however, striking, unless the patient is exhausted by long-continued pain and sleeplessness, or unless the aneurismal tumour is pressing upon the œsophagus or thoracic duct, and so interfering with nutrition.)

The following pressure symptoms and signs.—Lividity; œdema of the face, neck, and upper extremities; engorgement of the superficial veins of the head, neck, and upper extremities—are frequently observed in both cases (aneurism and tumour), but are much more common in tumour.

Pain is very much more prominent in aneurism than in tumour, and in many cases is one of the most important points of distinction between the two conditions. The pain, which occurs in cases of tumour is, as a rule, temporary, and is often due to associated pleurisy.

Cough seems to be somewhat more frequent in tumour.

Hæmoptysis is much more frequent in tumour than in aneurism, and this is particularly the case in the earlier stages of the disease.

Pressure on the recurrent laryngeal nerve, with its resulting symptoms, is more frequent in tumour than in aneurism, if we except aneurisms of the transverse portion of the aortic arch, which is the condition above all others, in which the left recurrent laryngeal nerve is implicated.

Pressure on the œsophagus, with resulting dysphagia, is more frequently the result of tumour than of aneurism.¹

Differences in the two radial pulses are more frequently observed in aneurism than in tumour.

Pressure on the sympathetic, with resulting changes in the pupil and vascularity on one side of the head and neck, seems to be more frequent in aneurism, but is often observed in tumour.

Lateral displacement of the heart is much more common in tumour.

Physical Signs at the Seat of the Tumour.—Inspection.—Prominence of the chest wall is very much more common in aneurism. Aneurismal prominence is usually localised, with a broad base and a well-marked apex, whereas a prominence due to tumour is almost always a general bulging of the chest wall. Pulsation over the prominence is rarely seen in the case of tumour. When it does occur, it is seldom localised, but is usually general, the whole chest wall appearing to be raised *en masse*. In aneurism, on the contrary, pulsation is common, and is usually localised to the prominence itself. The prominence due to an aneurism is more frequently

¹ Most of these statements of course refer to aneurisms and tumours as a whole; no attempt has been made to compare aneurisms of a special part of the aorta with tumours of a special part of the thorax.

situated in the neighbourhood of the second right interspace than in other parts of the thorax; the prominence due to a tumour rarely occupies this position. An engorged condition of the veins of the thoracic wall is more frequently seen in tumour than in aneurism. Œdema of the chest wall is more common in tumour than in aneurism. (The œdema which is due to aneurism is generally hard, and associated with tenderness on pressure, it is usually in fact, inflammatory; whereas, the œdema due to tumour is usually soft and painless, *i.e.* dropsical.) *Palpation.*—Aneurismal pulsation is frequently expansile, and often equals in intensity the impulse of the heart. The pulsation due to a tumour is not truly expansile; it is usually less defined than the pulsation due to aneurism; it seldom, if ever, equals in intensity the cardiac impulse, but is usually feeble and diffused over an extensive area of the chest wall. The vocal fremitus is often present over the dull area of a tumour, but is absent over the dull area of an aneurism.

Percussion.—The dulness due to aneurism is generally more localised, and more closely related to the course of the aorta than the dulness due to tumour. This point is one of great practical importance. *Auscultation* often gives very valuable information. In cases of tumour the breath-sounds can generally be heard over the position of the dulness, usually in the form of bronchial or tubular breathing, and the vocal resonance is usually present and often increased. In cases of aneurism, the breath-sounds and vocal resonance are not, as a rule, audible over the area of dulness. An exception to this statement occurs in those rare cases of thoracic aneurism in which the aneurismal sac is filled with laminated fibrine, and is in close contact with the trachea or large bronchi. The following is a case in point:—

Case of Aneurism of the Aorta, Innominate, Left Common Carotid, Right Common Carotid, and Left Subclavian, Arteries, simulating Solid Intra-thoracic Tumour.

M. F., æt. 64, a striker, was admitted to the Newcastle-on-Tyne Infirmary under my care on 18th February 1878, suffering from œdema of the face, neck, and upper extremities, and complaining of shortness of breath on the least exertion.

Previous History.—The patient, who had been under treatment for some days as an out-patient, stated that he had been through life an unusually strong healthy man. He had neither suffered from rheumatism nor syphilis. His present illness commenced some six weeks previous to admission, with shortness of breath on exertion. This was followed by swelling of the face and hands. He had of late occasionally experienced a slight pain under the manubrium sterni and in each shoulder. He had lost flesh, his voice had become 'thicker' than it used to be, and there had been some dysphagia.

Condition on Admission.—The patient was extremely dirty, the skin being of a deep brown colour; this was so marked that the case was sent to me as one of Addison's disease. The nipples, genitals, and axillæ, were not specially pigmented. The patient was fairly nourished, though he stated that he had lost flesh. The base of the neck was very much swollen and hard. The face, upper extremities, and upper part of the thoracic wall were œdematous. The superficial veins of the thorax and abdomen were engorged and prominent. (The veins of the head, neck, and upper extremities were also engorged, but were in great part hidden by the œdema.) There was well-marked præcordial vascularity.

The lips were swollen and blue.

The slightest exertion, such as getting out of bed to urinate, caused great shortness of breath. When at rest the breathing was natural. Food seemed to stick at a point corresponding to the manubrium sterni. There was some cough and frothy expectoration. The pupils were equal and contracted. The larynx was not examined. The radial pulses were equal both in time and volume. The carotid pulses seemed equal, but the pulsation in these vessels was difficult to feel owing to the brawny œdema of the neck.

The heart was of natural size; its valves healthy; its action weak.

There was well-marked percussion dullness over the manubrium sterni, and on each side of it especially to the left. On auscultation, tubular breathing and greatly increased vocal resonance were heard under the right sterno-clavicular articulation. Over the manubrium the breath-sounds were indistinct. The heart sounds were very faintly and distantly heard over the same part. Under the left sterno-clavicular articulation, and to the left of it, the breathing was bronchial. Posteriorly, the respiratory murmur was weaker in the right than in the left lung. Bronchial râles were heard here and there over the chest. There was no glandular enlargement. The blood was normal, except that the adhesiveness of the red globules was increased.

Diagnosis.—The diagnosis was an intra-thoracic tumour. It was thought to be solid because of:—

- 1st. The marked venous engorgement and local œdema.
- 2d. The absence of any decided pain.
- 3d. The somewhat extensive character of the dullness.

4th. The tubular breathing and increased vocal resonance under the right clavicle.

5th. The absence of any marked vascular sounds over the dull area.

6th. The non-accentuation of the aortic second sound at the base of the heart.

The treatment consisted in rest in bed and the administration of full doses of iodide of potassium.

Progress of the Case.—On 27th February the patient was decidedly better, the œdema was disappearing.

On 6th March, the œdema having almost gone, the thorax was again carefully examined, and important alterations were observed. The heart's action was considerably stronger than before. Well-marked pulsation could be felt in the supra-sternal notch over the manubrium sterni, the heart sounds could be distinctly heard, and they presented that toneless superficial character which is rightly considered as characteristic of an aneurism. The radial pulses were equal both in time and volume; a sphygmographic tracing showed nothing of importance, the only difference being that the percussion stroke was slightly shorter in the right than in the left. (See figs. 295 and 296.) (This is a point of some importance, for a good-sized aneurism of the innominate was found after death. The case does not, however, disprove Dr Mahomed's observations as to the diagnosis of innominate aneurism by means of the sphygmograph, for the sac of the aneurism was so filled with coagulum that the normal diameter of the blood channel was accurately preserved.)

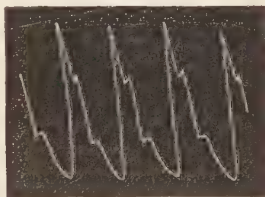
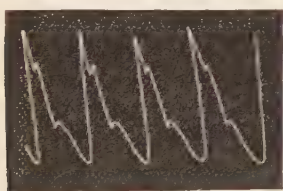


FIG. 295.—(Right radial.) Pressure 3 oz. FIG. 296.—(Left radial.) Pressure 3 oz.
FIG. 295 and 296.—*Case of Multiple-Aneurisms.*—M.F., æt. 64, admitted to the Newcastle Infirmary 18th February 1878, suffering from symptoms of intra-thoracic pressure. Died 10th March. *Post-mortem* showed dilatation and small aneurism of aortic arch. Fusiform aneurisms of the innominate, left common carotid and left subclavian arteries, just above their origins. The aneurisms were filled with firm clots, through which a straight narrow channel for the blood remained.

On 8th March the patient was worse; the œdema had returned. At the time of my visit he was seized with a severe rigor, during which the temperature rose to $105^{\circ}\frac{2}{2}$ Fahr.

On 9th March he spat up some muco-purulent matter mixed with blood. The base of the neck was very hard and brawny, and the skin had a red, erythematous appearance.

On 10th March he died.

The *post-mortem* was made twenty-seven hours after death. The base of the neck was much swollen and infiltrated with serum containing leucocytes.

The aorta, from its origin to the termination of the transverse portion of the aortic arch, was very much dilated and atheromatous. (Specimen exhibited at May meeting of the Edinburgh Medico-Chirurgical Society.) A sacculated aneurism, the size of a small cherry, projected from the anterior surface of the aortic arch. This small aneurism, to the outer surface of which the lung was firmly adherent, was completely filled with firm decolorised clot.

The innominate artery was irregularly dilated to the size of a bantam's egg. The vessel, at its origin and at its termination was of normal calibre. The sac of the aneurism pressed upon and was adherent to the trachea. The left common carotid artery, immediately after rising from the aortic arch, was uniformly dilated to the size of a damson. The right common carotid, immediately after its origin from the innominate, was dilated to the size of a small cherry. The left subclavian, immediately above its origin from the aorta, was slightly dilated.

Firm coagula channelled in their centres filled the aneurisms of the innominate, left common carotid, right common carotid, and left subclavian arteries. (See figs. 293 and 294.) The channels in the coagula exactly corresponded in their calibre to the normal size of these vessels.

The superior vena cava was pressed upon and greatly obstructed by the dilated arch. The heart was normal, its weight 10 oz. The other arteries throughout the body were natural. Both lungs were adherent at their apices. The upper lobe of the left lung was in places consolidated owing to the deposit of small masses of black pigment. The trachea and bronchi were inflamed. The other organs were normal.

The cause of the aneurismal dilatation of the arch and its branches was not ascertained.

The character of the heart and aortic sounds over the dull area of a tumour varies in different cases. In some, the cardiac or aortic sounds are inaudible; more frequently, both cardiac sounds are heard, free from murmur and weaker and more distant than over the heart itself; in a third group of cases a systolic murmur is audible, it is sometimes followed by a second sound, which is not, however, accentuated. In many cases the second sound is faint or absent. The auscultatory phenomena which are heard over an aneurismal sac have been fully detailed, and need not again be described. Let me repeat, however, that a murmur is more frequently

absent than present in those cases in which the cardiac valves are healthy;¹ and that an accentuated aortic second sound, with or without a systolic murmur, is of great diagnostic value, when other symptoms and signs of aneurism are present.

Associated diseased conditions.—Cardiac valvular lesions (more especially aortic regurgitation) and general atheroma are frequently present in aneurism, but comparatively rare in tumour. Pulmonary lesions, pleurisy, glandular enlargements, and new growths in the liver and other internal organs are often associated with intra-thoracic (cancerous and sarcomatous) tumours. The associated diseased conditions and the general pathological tendencies of the individual are therefore of the greatest importance from a diagnostic point of view.

The progress of the case and the results of treatment.—Intra-thoracic tumours (cancerous and sarcomatous growths) steadily, and, as a rule, rapidly progress from bad to worse, and are not amenable to treatment, though Walshe has seen temporary improvement in some cases of cancer.² The progress of aneurism is, as a rule, much more gradual, and the symptoms can, in most cases, be temporarily relieved by treatment (rest, dieting, and iodide of potassium).

Step No. 2. If an aneurism is present, is it an aneurism of the thoracic aorta?

Aneurisms of the transverse portion of the aortic arch are, in many cases, with difficulty distinguished from *aneurisms of the innominate artery*. The distinction is a matter of practical importance, for surgical treatment, which is justifiable and advisable in many cases of innominate aneurism, is hardly to be recommended when the aneurism is aortic. In many cases the aneurismal tumour involves the transverse portion of the aortic arch and the innominate artery; and it is more

¹ In speaking of the value of a systolic murmur in cases of this description Stokes says:—‘If we suppose a case in which the evidences on both sides were nearly balanced, the existence of a single soft systolic bellows murmur should incline our opinion towards cancer. Bellows murmur in aneurism of the arch is a more rare circumstance than has been supposed.’—*Diseases of the Heart and Aorta*, p. 605.

² *Diseases of the Heart*, p. 579.

especially in those cases in which the symptoms and signs of innominate aneurism are present, and in which the question of operative procedure therefore arises, that it is important to determine whether the aneurismal tumour involves the aortic arch or not. In many cases the decision is easy, in others difficult, in some cases impossible.

The question is to be decided chiefly by *careful percussion of the aortic arch*. Attention must also be directed to the following points:—

1. *The position of the aneurism.*—Innominate aneurisms are usually situated to the right of the middle line, and extend chiefly upwards into the neck. The innominate artery rises, it will be remembered, on the level of the upper edge of the first interspace, or that of the lower part of the first cartilage, behind the right half of the upper sternal region, passes upwards and to the right, bifurcating to the right of the trachea, or a little above and to the right of the sterno-clavicular joint.¹ When, therefore, the aneurismal tumour is situated under the right sterno-clavicular articulation, and when percussion fails to demonstrate any distinct enlargement of the aortic arch, the aneurism is probably innominate.

2. *The effects of pressure on the right common carotid and right subclavian arteries.*—Firm pressure on the vessels arising from the innominate trunk arrests, it is said, or materially lessens the pulsation of an innominate aneurism, but fails to produce any material effect upon aneurisms of the aortic arch. This and the following points are, however, of little value in deciding whether, in cases of innominate aneurism, the sac involves the arch of the aorta or not.

3. *The condition of the pulse.*—Aneurisms of the innominate are much more likely to modify the characters of the right radial pulse (in the manner described on p. 283) than aneurisms of the aortic arch.

4. *The nature of the pressure symptoms.*—Aneurisms of the innominate, which rarely extend to the right of the middle line, are much less likely to produce pressure upon the trachea, œsophagus, and left recurrent laryngeal nerve but are more

¹ Walshe, *Diseases of the Heart*, p. 6.

likely to press upon the strands of the right brachial plexus, and upon the right recurrent laryngeal nerve, than aneurisms of the aortic arch.

Prognosis.—Aneurisms of the thoracic aorta almost invariably prove fatal, the few cures which have been met with, being amongst the curiosities of medicine; the prognosis, as regards the ultimate result, is, therefore, most unfavourable. The duration is a matter of the greatest uncertainty, for there is always a risk of rupture and of sudden death. Our opinion, as to the probable duration of the case, must be guided to some extent by (*a*) the stage of the disease, (*b*) the position of the aneurism, and (*c*) the circumstances, surroundings, and mental temperament of the patient. The larger the sac, other things being equal, the shorter the probable duration of the case. Aneurisms of the sinuses of Valsalva probably kill sooner than aneurisms of any other part of the aorta. Aneurisms of the ascending portion of the arch, above the sinuses of Valsalva, which make their way forwards, have, on the average, the longest duration. Aneurisms of the transverse and descending portions of the aortic arch, and of the descending thoracic aorta, probably occupy (as regards duration) a mid-position, *i.e.* they usually last longer than aneurisms of the sinuses of Valsalva, but shorter than aneurisms of the ascending portion of the arch.

When the sac is pressing upon the trachea, upon a main bronchus, or upon the œsophagus, the fatal termination is, as a rule, close at hand.

Patients who are obliged to exert themselves in order to gain a livelihood, more particularly those who have to follow laborious occupations, are at a serious disadvantage, for rest and the avoidance of all strain are the most important means of treatment. Persons of an irritable disposition, those who are easily excited, who will not submit to the restraints of treatment, and those who indulge in alcoholic excesses, are also heavily handicapped; in them the average duration of the disease is considerably shorter than in other people.

The associated pathological conditions must also, of course, be taken into account in forming an opinion as to the probable duration of the case.

Treatment.—The great object of treatment in the case of all aneurisms is to endeavour to effect a cure by causing solidification or contraction of the sac.

In the case of aneurisms of the thoracic aorta, surgical means of cure are, with the exception of galvano-puncture, which will be presently considered in detail, inadmissible. Reliance must, therefore, be chiefly placed upon general measures and the use of drugs.

The *first* indication is to keep the circulation as tranquil as possible. In carrying out this indication, the intelligent co-operation of the patient is essential. It is necessary, therefore, to explain to him the nature of the disease, the things which are to be avoided, and the exact objects which we have in view.

In the *first* place, all bodily exertion and mental excitement, which increase the intra-arterial blood pressure, are to be avoided. The ideal treatment is to keep the patient at absolute rest in bed; but since this treatment to be at all effectual must be of long duration, patients will not, or cannot, with rare exceptions, submit to it. Absolute rest in bed undoubtedly gives the patient the best chance, but it is doubtful if many actual cures would be effected even if this indication could always be strictly carried out. Whenever the mental temperament and circumstances of the patient will permit, the ideal treatment should be enforced. When the patient will not or cannot lie in bed, the amount of muscular action should be as small as possible, in particular, sudden efforts or strains are to be avoided.

In the *second* place, all causes of mental excitement, especially of *sudden* mental excitement, are to be guarded against.

In the *third* place, the diet must be strictly regulated. The amount of food allowed should be as small as is compatible with the proper nutrition of the body and the maintenance of the coagulating properties of the blood; it must be easily

digested, but at the same time nutritious. The amount of liquid must also be restricted, and alcoholic stimulants absolutely forbidden. Mr Tufnell, whose treatment of thoracic aneurisms by rest and low diet has been so successful, limits the amount of food to :—2 oz. of bread and butter and 2 oz. of new milk for breakfast ; 2 or 3 oz. of bread, and 2 or 3 oz. of meat for dinner, with 2 to 4 oz. of milk or claret ; 2 oz. of bread and 2 oz. of milk for supper. Personally I have never enforced such a very restricted diet. I always, however, impress the patient with the importance of strict moderation both as regards food and drink.

In the *fourth* place, the condition of the bowels must be carefully regulated, so that all straining at stool is avoided. Sexual intercourse must of course be forbidden.

In the *fifth* place, drugs which reduce the arterial tension, and which experience has found useful, are to be given. Iodide of potassium, is by far the most valuable remedy. When the case first comes under observation, and more especially when pain is a prominent symptom, the drug should be given in full doses (30 grs. three times a day) ; as soon as the pain subsides the dose may be reduced, though personally I have been in the habit of continuing the full dose so long as it is satisfactorily borne. Dr George Balfour, who has done so much to establish the iodide plan of treatment in this country, thinks that the large doses which he formerly recommended are unnecessary ; he now gives smaller doses. The proper dose for each case is, he states, that which will lower the blood pressure without increasing the frequency of the cardiac contractions. In speaking of the manner in which the dose which is suitable for each case is to be ascertained, he states :—‘ Accordingly, those cases which came next under treatment were put to bed for a few days without further treatment, their pulse-rate being carefully taken night and morning. So soon as the average pulse-rate in recumbency had been sufficiently ascertained, 10 grs. of iodide of potassium in some bitter infusion, usually chiretta, were given three times a day. If the pulse-rate remained unchanged, the dose was increased to 15 grs. three times a day, and

we have not yet been able to get beyond this dose ; while very often we have not been able to give more than 10 grs. without raising the pulse-rate.¹

The exact method in which the iodide of potassium acts is doubtful. Some authorities think that it produces some modification in the blood, which favours coagulation and clotting within the sac. Dr Balfour thinks that it acts 'mainly by some peculiar action on the fibrous tissue, whereby the walls of the sac are thickened and contracted, while if coagulation should take place within the sac, it plays but a very secondary and unimportant part, depends for its occurrence solely on the remora of the blood, and is in no respect due to the iodide of potassium.'² He states that 'hypertrophy of the muscular coat, where that still exists, and of the adventitia, with concomitant contraction, are found in all aneurisms which have been treated with the iodide with any measure of success.'³ My own observations lead me to doubt the occurrence of hypertrophy of the muscular coat in the wall of the sac. I believe that the iodide acts chiefly by reducing the blood pressure and relieving the tension within the sac—a point which Dr Balfour also thinks is of the greatest importance—and partly, perhaps, by removing the endarteritis obliterans, which is often present, more especially in syphilitic cases, in the minute arteries which ramify in the walls of the sac and which supply it with nutrient fluid. The administration of small doses of chloral (7 grains three times a day) has appeared to me beneficial in some cases in which the arterial tension is distinctly increased. Possibly nitroglycerine might be advantageous in cases of this description. The external application of cold is the best means of relieving temporary overaction of the heart. Aconite is also recommended by some writers. Both means are at the best, palliatives, and produce no permanent benefit. In those cases in which there is a tendency to cardiac excitement, our main object should be to ascertain the cause of the over action and to remove it ; and in this connection it is important to remember that starvation or anything else which causes anæmia, produces an irritable condition of the heart, in which

¹ *Diseases of the Heart*, p. 458. ² *Ibid.*, second edition, p. 454. ³ *Ibid.* p. 457.

palpitation is readily excited by mental agitation or slight bodily exertion. Care must, therefore, be taken that while the diet is restricted, it is not reduced to such a point as will produce anæmia and cardiac irritability.

The hypodermic injection of ergotine was recommended by Langenbeck some years ago as a means of producing contraction of the sac; but subsequent observers have not met with the same favourable results which he described.

It has been proposed to produce coagulation within the sac by mechanical means, such as ligature of the great vessels arising from the aortic arch, the introduction of iron wire or horse hair, or the injection of perchloride of iron into the sac, and by galvano-puncture. The results of ligaturing the vessels arising from the sac are not favourable. Surgical interference of this sort is, in my opinion, seldom justifiable, and so far as I can form a judgment, inferior to galvano-puncture. The introduction of iron wire or horse hair into the sac, and the injection of perchloride of iron or other astringents should never be attempted.

Galvano-puncture.—This method of treatment should only be attempted when, after a fair and prolonged trial, the treatment by iodide of potassium, rest, and dieting, has failed, or, in those cases in which the sac is obviously on the point of rupturing, and in which it is, therefore, necessary to have recourse to some plan of treatment calculated to produce immediate relief. Galvano-puncture may be performed with two distinct objects, viz. :—

(1) To coagulate the whole contents of the sac at one sitting.

(2) To form a small, firm clot, which will act as a nucleus on which layers of coagula will be subsequently deposited.

For the successful performance of galvano-puncture it is necessary to use a current of considerable strength. Some writers recommend the Leclanché element, which is so convenient for many purposes, but the Stöhrer's element is probably, as Dr de Watteville points out, better suited for electrolysis.¹ The *needles* must be well insulated, and there

¹ *Medical Electricity*, second edition, pp. 67 and 200.

must be no shoulder where the insulating material joins the barb. Authorities differ as to whether one or both needles should be introduced into the sac. It is probably unnecessary to introduce a needle connected with the negative pole into the sac; one or more needles should, therefore, be introduced and connected with the positive pole, and the negative pole connected with a large flat copper electrode (covered with wash-leather) placed on the abdomen. Dr de Watteville states that 'in order to avoid the burning sensation and vesication of the skin, a layer of modeller's clay may be effectually placed between the plate and skin, as suggested by Dr Apostoli of Paris.'² Before the needle or needles are introduced into the sac, the physician must see that the battery is in working order; this is best tested by observing the coagulating power of the current on albumen. (The white of an egg is to be placed in a saucer, and the needles, connected with the battery, inserted in it. On the passage of the current a small firm clot is produced at the positive, and a large, loose, whipped-up, frothy clot, around the negative needle.) The exact nature of the procedure must be explained to the patient, and he must be cautioned not to make any sudden movement when the needle is being introduced. The sensibility of the skin may be deadened at the seat of the proposed puncture, by the application of ice or ether spray, but it is not desirable to freeze the skin thoroughly, lest its vitality should be impaired and the rupture of the sac hastened. The needles, which should be very sharp, must be slowly but steadily introduced into the sac. After the operator is satisfied that the insulating material is well through the wall of the sac, the needle may be connected with the battery.

The duration of the *séance* varies with the object of treatment. When it is desired to coagulate the whole contents of the sac, the operation must be continued until the sac is felt to be firm and solid. Two or three hours may be required for this purpose. When, on the other hand, the object is to produce a small, firm coagulum, the current should not be passed through the sac for more than twenty minutes or half

¹ *Medical Electricity*, second edition, p. 199.

an hour. Should bleeding, or any other untoward symptom occur during the course of the operation, the current should be at once 'broken' and the needles withdrawn. The needles are best withdrawn by means of a slow rotatory movement. A pad of lint must then be applied over the point of puncture.

It is usually necessary to repeat the galvano-puncture more than once. Between the *séances* the treatment by rest, iodide of potassium, and restricted diet, must be steadily persevered with. The operation is apt to be followed by inflammation of the sac wall. This is the great danger. The risks of bleeding (either during the operation or after the withdrawal of the needles) and of embolism are extremely slight.

Galvano-puncture is, I repeat, chiefly useful as a last resource. It should only be attempted in those cases in which (1) a prolonged and patient trial has been given to rest, diet, and iodide of potassium, and in which that method has failed; or (2) in those cases in which the aneurism is progressing rapidly or is on the point of rupturing, and in which, therefore, it is necessary at all hazards to arrest the progress of the disease without delay. The treatment is most likely to be beneficial in those cases in which the aneurism is distinctly sacculated, and more especially when the chest wall has become perforated, and an external false aneurism has been formed. Galvano-puncture is not advisable in those cases in which aneurismal dilatation is general, globular, or fusiform.

For the relief of urgent symptoms other measures may of course be employed.

Pain, which is such a prominent symptom in many cases of aortic aneurism, is best relieved by the administration of large doses of iodide of potassium. In some cases, it is necessary to give hypodermic injections of morphia, but the systematic administration of this drug is seldom required except during the first few days of treatment, *i.e.* when the patient first comes under observation, and before the iodide has had time to act.

Dyspnœa, *dysphagia*, *hæmoptysis*, and other prominent symptoms, must be met by appropriate remedies. I need not go into details, further than to say, that attention to the

mechanical position of the parts and the administration of such remedies as iodide of potassium, the external application of cold to the surface of the tumour, and subcutaneous injections of ergotine, are the most likely means of giving relief. When the dyspnœa is very severe, and clearly laryngeal in character, the question of tracheotomy has to be considered. The operation should only be performed when the observer is satisfied that the dyspnœa is due to laryngeal paralysis or spasm. It is of course quite useless and inadmissible in those cases in which the dyspnœa is due to compression of the trachea or bronchi. Bristowe's opinion—that the paroxysmal attacks of dyspnœa, which many observers ascribe to spasms or paralysis of the glottis, in reality depend upon the pressure of the sac on the trachea—must be remembered.

When the sac threatens to rupture externally, galvanopuncture is, as I have previously mentioned, advisable. Should it fail, an attempt may be made to prevent rupture by supporting the tumour externally by means of a pad of felt, tin, or other material.

COARCTATION OF THE THORACIC AORTA.

Ætiology and Pathology.—Cases are occasionally met with, but none have come under my own observation, in which the thoracic aorta is constricted just beyond the point at which it is joined by the ductus arteriosus Botalli.

The constriction seems to be due to the fact that the obliterative process, which closes the ductus arteriosus soon after birth, passes to, and involves the part of the aorta to which the ductus arteriosus is attached. In some cases, the constriction is slight; in others great; occasionally complete.

Pathological physiology.—The effect of the constriction is, of course, to interfere with the free passage of the blood from the transverse into the descending portions of the aortic arch. In those cases in which the constriction is considerable or complete, the descending portion of the thoracic and the abdominal aorta and their branches have to be supplied in a circuitous or roundabout manner; the innominate and left subclavian arteries, the deep-seated arteries of

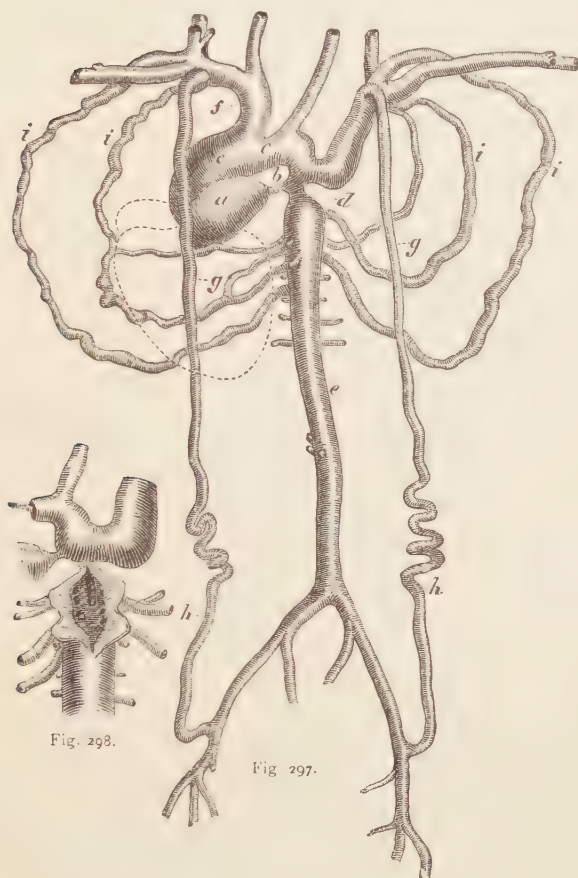


Fig. 298.

Fig. 297.

(From Walshe, after an unpublished drawing by Carswell.)

FIG. 297. *a*, pulmonary artery; *b*, arterial duct; *c*, arch of aorta; *d*, coarctation of aorta; *e*, descending aorta; *f*, innominate artery; *g*, internal mammary artery; *h*, epigastric artery; *i, i, i, i*, deep-seated arteries of neck and intercostals, forming, together with the internal mammary and epigastric arteries, a collateral circulation with the thoracic and abdominal aorta and internal iliacs.

FIG. 298. Aorta laid open, showing by probe the amount of constriction.

the neck, the superior intercostal, internal mammary, and epigastric arteries become enormously enlarged, and the circulation is carried on through the inosculation which are so well represented in figs. 297 and 298.

Symptoms.—In those cases in which the constriction is slight, there may be no symptoms. When the constriction is great, dyspnœa, cough, hæmoptysis, and vertigo, are the chief symptoms which have been observed.

Physical signs.—The passage of the blood through the constricted portion of the aorta generates a murmur, which is post-systolic rather than systolic, is extensively propagated down the course of the aorta, and does not exactly correspond to any of the ordinary systolic murmurs generated within the heart.

The most characteristic indication of the condition is, however, the enlargement of the vessels arising from the aortic arch, and of the intercostal and epigastric arteries, through which the anastamotic circulation is carried on; in consequence of this enlargement, pulsation, which is sometimes so great as to suggest the presence of an aneurism of the innominate or transverse portion of the aortic arch, is observed at the root of the neck. 'Occasionally,' according to Walshe, 'local expansile impulse, aneurismal to the feel (and sometimes strong enough to gradually wear away the ribs), may be felt from place to place in the latter (intercostal) vessels.'¹ Thrills and murmurs can usually be heard over the enlarged arteries.

Diagnosis.—The condition has seldom been suspected, much less recognised, during life. The slighter forms of constriction cannot be positively diagnosed. When the constriction is great, and when enlargement of the intercostal and epigastric arteries can be recognised during life, the diagnosis would be easy. In those cases in which the great vessels arising from the arch of the aorta are much dilated, aneurism may be simulated.

Prognosis.—A slight amount of constriction is not incompatible with a long life; the prognosis in the more severe forms is very uncertain; in some cases the patient dies

¹ *Diseases of the Heart*, p. 536.

suddenly from rupture of the aorta, or one of its dilated branches; in others, in consequence of the secondary alterations, such as cardiac dilatation, which are produced in the heart, or parts of the circulation behind the left heart (*i.e.* the lungs, right heart, etc.); in others again, from acute inflammatory changes in the heart or aorta.

Treatment.—The main objects of treatment are to keep the circulation as quiet as possible, and to avoid exposure to cold and other conditions likely to induce acute inflammatory changes in the heart and aorta. When symptoms of mechanical derangement of the circulation arise, they must be treated in accordance with their nature and the special indications in each case.

For further details of this interesting but rare condition the reader is referred to Dr Walshe's description,¹ to which I am largely indebted for the foregoing account of the disease.

¹ *Diseases of the Heart*, p. 533.

APPENDIX.

THE EXAMINATION OF THE HEART BY MEANS OF THE CARDIOGRAPH.

By means of the cardiograph it is possible, in many cases, to obtain a graphic record of the cardiac impulse; and the information, which this method of examination affords, is sometimes of considerable diagnostic value.¹

Cardiographic tracings are usually obtained from the pulsations of the left apex-beat, for the cardiac impulse is, as a rule, better defined at this point on the surface of the chest than at any other; and since the apex-beat (*i.e.* the left apex-beat) is due to the impulse of the ventricles, more especially of the left ventricle, against the chest wall, it follows that tracings taken from the apex-beat represent the alterations which take place in the ventricles (more especially in the left ventricle) during the cardiac cycle.

The information which may be derived from the cardiograph. By means of the cardiograph we are able, in some cases of cardiac disease (but not in all) to obtain a graphic record of the condition of the ventricles (more especially of the left ventricle) during their systole and diastole, and to determine:—

- (1.) The relative duration of the ventricular systole and diastole.
- (2.) The manner in which the ventricular systole is being carried out.
- (3.) The manner in which the ventricles are being filled with blood during their diastole.

¹ The description of the cardiograph has not been included in the text, for the instrument is rarely used even in hospital practice, and cannot, as yet, be said to be one of the ordinary means of clinical investigation.

Since the filling of the ventricles to a large extent depends upon the condition of the auriculo-ventricular orifices and of the auricular contractions (*i.e.* of the muscular wall of the auricles), the cardiograph affords in some cases :—

(4.) Information as to the condition of these parts (more especially of the condition of the mitral orifice and the muscular wall of the left auricle).

Further, by means of the cardiograph it is possible to clear up some obscure cases of cardiac disease, and to ascertain the exact relationship of murmurs (which it is difficult or impossible to 'time' in any other way) to the different periods of the cardiac cycle.

By comparing tracings taken from successive beats of the heart (subject to the precautions which will be presently described), the presence of inequalities and irregularities in the cardiac action can be graphically demonstrated.

The cardiograph is, however, in many cases an unsatisfactory instrument to work with,—much less satisfactory than the sphygmograph. The button of the sphygmograph can, with rare exceptions, be speedily and easily applied so as to exert direct pressure upon the radial artery, and the tracing which is thus obtained is an accurate representation of the alterations which take place in the vessel,¹ but it is often difficult or impossible to obtain a cardiographic tracing, for the apex-beat is sometimes very feeble, or altogether effaced; the tracings which are obtained are often inverted, and therefore unreliable. Even good tracings are difficult to analyse, for they represent not only the alterations in the size and state of the ventricles (more especially of the left ventricle) which result from the contraction and relaxation of their muscular walls, and from the collapse which attends the emptying, and the distention which results from the filling of their cavities; but also the movements of the heart as a whole, for every alteration in the heart (or the surrounding parts) which pushes

¹ The radial artery at the lower end of the radius is quite superficial, and rests upon bone; in most cases, therefore, it can be directly compressed between the button of the sphygmograph and the flat surface of the radius on which it lies.

it against, or causes it to recede from, the front wall of the chest, produces alterations in the character of the cardiac impulse. The force and character of the apex pulsation depend in fact, to a very large extent, upon the condition of the anterior margin of the left lung (whether distended during inspiration or retracted during expiration); while the movements of the chest wall which attend inspiration and expiration, and the influence, which the acts of inspiration and expiration have on the blood-pressure within the cardiac cavities, are actively manifested in the cardiographic tracing. It must be remembered then, that cardiographic tracings do not represent in such a simple and accurate manner the alterations which are taking place within the heart, as sphygmographic tracings represent the alterations which are going on within the radial artery.

FORMS OF CARDIOGRAPHS.

1. *Galabin's Cardiograph* (see fig. 299), which probably gives the most reliable tracings, is simply a modification of Marey's Sphygmograph.

Dr Galabin's description of it is as follows¹:—‘The brass frame of the instrument resembles that of the sphygmograph, except as regards the bar which carries the knife edge, A, through which the motion is transmitted to the long lever. This bar, B, is made up of two parts, of which one slides within the other, and can be fixed by means of a screw, C, in whatever position is desired. There is also a second knife edge, D, which can be raised or lowered at pleasure, attached to the same bar at a greater distance from the axis of the long lever. By this means the magnifying power of the instrument, as regards the vertical height of the curve described, can be varied from ten to about a hundred. The brass frame, which in the sphygmograph is rigidly fixed to two parallel bars of ivory by which it is supported, is freely suspended in the cardiograph by means of two transverse rods of steel, E. These are attached by joints, F, which allow both of vertical and horizontal adjustment, to four vertical rods of

¹ *Medico-Chirurgical Transactions*, vol. lviii. p. 359.

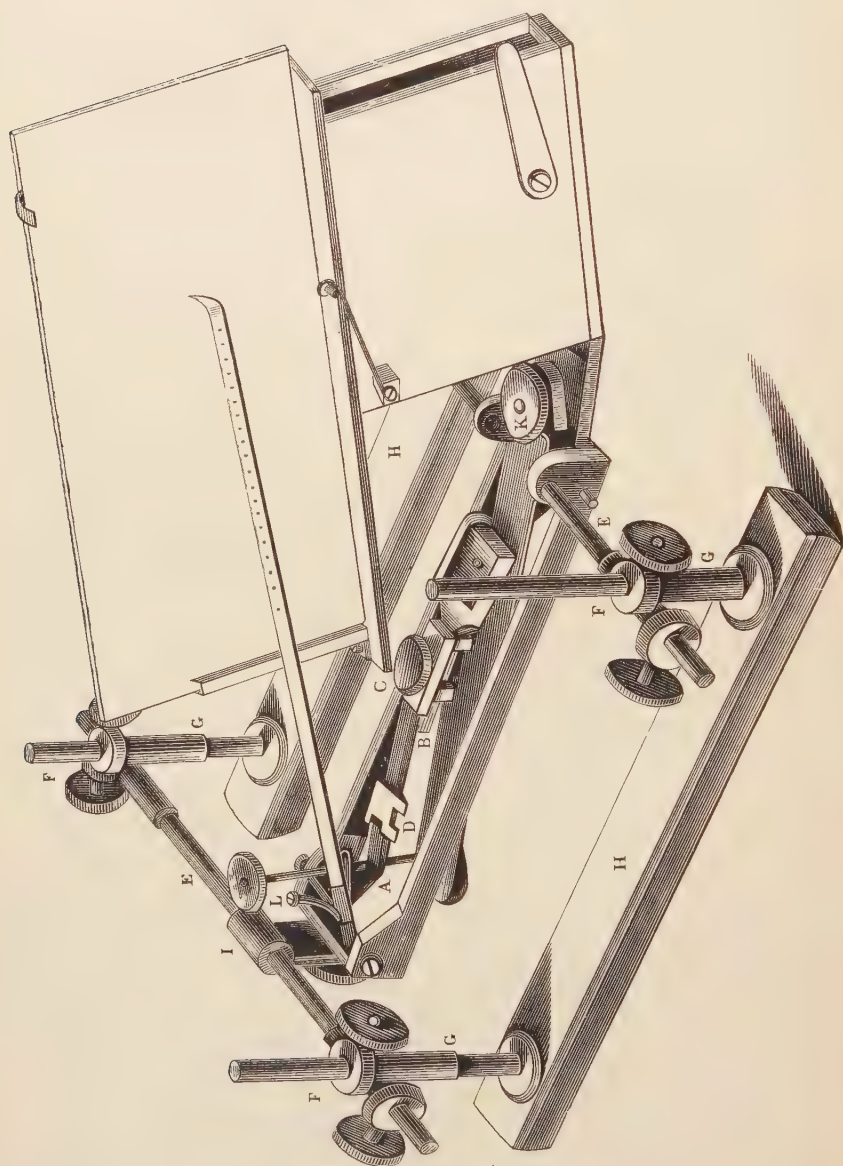


FIG. 299.—Galabin's Cardiograph. The description of the letters is given in the text. Copied from the *Medico-Chirurgical*

steel, G, each pair of which is inserted into a bar of wood covered with leather,—by means of these wooden bars the instrument rests upon the chest. They can be separated to a width of nearly five inches, and the instrument can be raised or lowered at pleasure at either end, and in this way it can be adapted to a chest of any size or shape.

‘In order that vertical adjustments at either end may be possible independently, the brass frame is not in immediate contact with both the transverse bars which support it, but at one end it is suspended by an intermediate piece of brass, I, which, when the instrument is in position, is tightened and made rigid by a screw. The spring which is employed to press upon the centre of impulse is arranged in a mode similar to that adopted in the sphygmograph. The mechanism, however, by which the amount of pressure is finally adjusted is a simpler one than that employed in any one of the various forms of sphygmograph now generally used. This simplification is rendered possible by the fact that, in the case of cardiographic tracings, a knowledge of the exact amount of pressure employed would have little or no significance. The adjustment is effected by means of a screw, K, which perforates the short arm of the spring lever, B. The weight of the lever itself is also counterbalanced by a small antagonistic spring.

‘In this way the pressure upon the point at which the spring pad is applied can be reduced almost to zero, and thus it is easy to obtain with this instrument a tracing representing the backstroke in veins which even the weight of the spring of an ordinary sphygmograph is generally sufficient to extinguish.

‘There are also two small springs, L, of different strength, to depress the long lever and prevent its being jerked away by any sudden motion from the knife edge on which it rests. Either of these can be used or turned aside at pleasure. When the instrument is used in a vertical position it is generally better to dispense with this small spring, since it adds a little to the friction, and it is found that the lever does not become separated from the knife edge, provided that the magnifying power of the cardiograph be so adjusted that its movements

have only a very moderate amplitude. If, however, it is desired to take a tracing from a patient in a sitting or standing posture, it is necessary to use the secondary spring, for then the recording lever is no longer kept in position by its own gravity.

'The cardiograph may be fixed upon the chest by two narrow straps passed round the body and fastened by buckles. These should be partly elastic, that they may yield a little if the patient makes an inspiratory effort while the clockwork is in motion. In this way the disturbing influence of the muscular movement upon the cardiac curve is diminished. As soon, however, as the observer has acquired some dexterity, it will be found sufficient in most instances to hold the instrument against the chest with the hand.'

2. *Marey's Cardiograph and its modifications.*—Marey's cardiograph consists of two tambours connected by means of a flexible hollow tube. A button, which is attached to one

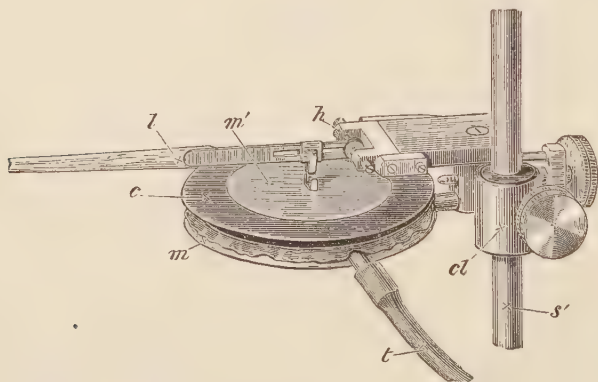


FIG. 300.—*Marey's Tambour.*

The metal chamber *m* is covered in an air-tight manner with the india-rubber *c*, bearing a thin metal plate *m'* to which is attached the lever *l* moving on the hinge *h*. The whole tambour can be placed by means of the clamp *cl* at any height on the upright *s'*. The india-rubber tube *t* serves to connect the interior of the tambour to which the lever is attached with the cavity of the tambour which is placed over the apex-beat.

of the tambours, is accurately applied over the position of the apex-beat; to the second tambour the recording lever is attached. (See fig. 300.) Any impulse communicated to the button of the first tambour drives the air out of it, and through the connecting-tube to the second tambour; the alterations which are produced in the second tambour in this manner, are communicated to the writing lever, which records the tracing on a piece of smoked paper attached to a revolving cylinder called a polygraph.

3. *The ordinary Sphygmograph.*—In some cases in which the apex-beat is very distinctly localised, cardiographic tracings may be obtained by means of the ordinary sphygmograph. In the majority of cases of cardiac disease the impulse is too diffused to permit of the satisfactory use of Marey's sphygmograph for this purpose, for the shock of the heart is communicated to the framework as well as to the button of the instrument and the tracing is consequently unreliable. Dr Sansom frequently uses Pond's sphygmograph as a cardiograph.

DIRECTIONS FOR TAKING A CARDIOGRAPHIC TRACING.

1. *Place the patient in the proper position.*—If possible he should be placed on his back in bed; unfortunately in many cases a cardiographic tracing cannot be obtained in this position, for when the cardiac impulse is feeble it may be necessary to make the patient sit up or lean forward in order to get an apex-impulse of sufficient power to produce a cardiographic tracing; again, in many cases of cardiac disease, the patient is unable, on account of shortness of breath, to lie in the recumbent position. When a cardiographic tracing is taken by means of Galabin's instrument with the patient in a sitting position, the small secondary spring (L) must be used in order to keep the writing lever in contact with the turned-up knife edge.

2. *Carefully ascertain and mark the exact position of the maximum point of pulsation of the apex-beat.*

3. *Apply the instrument, having previously screwed up the*

clockwork and smoked the paper and fitted in the slide which carries it.

The horizontal adjustments must be altered so that the wooden bars rest firmly on the chest-wall beyond the range of the cardiac impulse; the ivory pad must be accurately applied over *the exact point of maximum apex-pulsation*; the vertical adjustments being altered in order to permit of its accurate application to the wall of the chest; (the accurate application of the ivory pad to the exact point of maximum apex-pulsation is of the greatest practical importance, for unless this point is attended to, the tracing is apt to be inverted, and therefore unreliable); the sliding bar must be so adapted that a suitable amount of movement of the writing lever is obtained (after any alteration of the sliding bar to which the knife edge is attached, the ivory button must of course be again carefully reapplied over the point of maximum apex-pulsation); the instrument is then fixed to the chest by means of the straps (tracings may be obtained by holding the instrument in contact with the chest-wall, but it is, I think, advisable, for the sake of accuracy, to fix the instrument and not merely to hold it).

4. *Make the patient expire and hold his breath, and while he is holding his breath start the clockwork and take the tracing.*

This is also a point of the greatest practical importance, for, if the tracing is taken during the act of respiration it becomes complicated, the cardiac impulse being interfered with by the anterior edge of the left lung, and the movements of the chest wall which attend the act of respiration being communicated to the writing lever. It is of the utmost importance therefore to take the tracing *during complete expiration* (for then the anterior edge of the left lung is retracted, and a large part of the anterior surface of the heart is in direct contact with the chest wall), and *while the patient holds his breath* (in order that the movements of the lever may represent the cardiac impulse only, and not be complicated by the respiratory movements of the chest wall). It must also be remembered that the acts of inspiration and expiration materially modify the condition of the intra-cardiac circulation.

Unfortunately these ideal conditions cannot always be obtained. In many cases of cardiac disease the patient is suffering from dyspnœa, and is quite unable to hold his breath ; in others, again, the lungs are emphysematous, and during expiration the greater part, or even the whole of the anterior surface of the heart, is overlapped, and its impulse therefore obscured.

ANALYSIS OF A CARDIOGRAPHIC TRACING.

The analysis of cardiographic tracings is attended with great difficulties, and some important points are not yet clearly understood. The conclusions, which have been arrived at, have been partly drawn from observation (such as those of Chauveau and Marey) made by recording the pressure within the cardiac cavities of the lower animals, *e.g.* the horse ; partly from tracings taken from the exposed hearts of the lower animals, the button of the tambour being in direct contact with the exterior of the ventricles ; and partly from cardiographic tracings taken from the cardiac impulse as felt on the chest wall of men.

A cardiographic tracing consists of a series of curves (see fig. 301), each one of which corresponds to a complete cardiac revolution, *i.e.* the time which elapses from the commencement of one ventricular systole to the termination of



FIG. 301. *Normal Cardiographic tracing.*—(After Galabin.)

the ventricular diastole. Each individual cardiac curve (see fig. 302 A to B) is composed of two portions, 1 and 2, which represent the systole and diastole of the ventricles respectively ; and since cardiographic tracings in man are usually

taken from the left apex-beat, it is the systole and diastole of the left ventricle which is most accurately represented. In the following description I shall then, for the sake of simplicity, limit my remarks to the left heart.¹

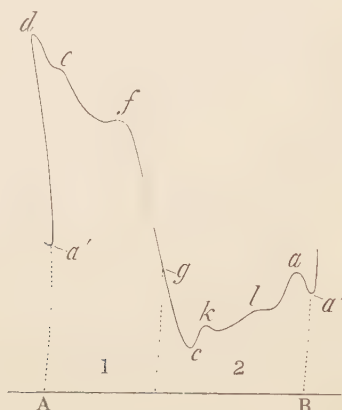


FIG. 302.—Normal cardiographic wave.—(Enlarged and modified from Galabin.)

A. B. base line; 1, systolic, and 2 diastolic portions of the tracing; for the significance of the other letters see text (p. 760, *et seq.*)

The systolic portion of the tracing.

The systolic portion of the tracing taken from the apex-impulse in man, may for descriptive purposes, be divided into the following parts:—

1. A line of ascent (*a'* to *d*).
2. A summit or apex (*d*).
3. A line of descent (*d* to *c*).

Professor Michael Foster concludes that the down-stroke from *d* to *a* (in fig. 303) corresponds to the relaxation of the

¹ In normal cardiographic tracings the *whole* of each individual cardiac curve or wave is not, as in the case of the normal sphygmographic or pulse curve, situated above the base line of the tracing, *i.e.* a line drawn through the commencement of the up-stroke of succeeding curves or waves; but the commencement of the up-stroke is situated above the portion of the cardiographic curve which represents the ventricular diastole (see fig. 301). The hyperdiastolic pulse curve presents the same character.

ventricles, and if this interpretation is to be applied to tracings taken from the apex-beat of man, the whole of the down-stroke ought to be included in the diastolic and not in the systolic portion of the tracing. But as Dr Galabin points out, the second sound in man is usually heard to occur towards the termination rather than at the commencement of the line of descent, and if this is so, and if, as Professor M. Foster points out, 'at the actual closure of the semilunar

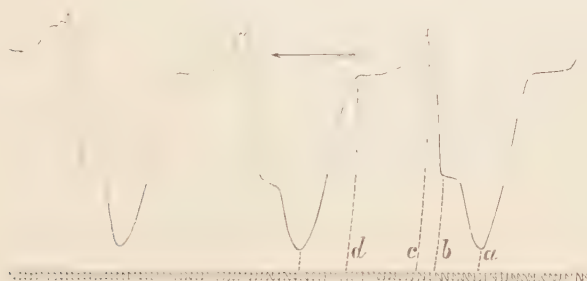


FIG. 303.—'Normal heart curve showing changes in the antero-posterior diameter of the ventricle obtained from the cat by a light recording lever moved by a button which pressed gently on the anterior surface of the ventricle. The time curve gives 50 double vibrations per second, and lines have been drawn to show the duration of the different phases of the ventricular movement. *a* to *b* corresponds to the distension of the ventricle including the auricular systole, the wave-like rise during this period being due to the increase in the diameter of the ventricle resulting from the entrance into it of the contents of the auricle. The period from *b* to *c* corresponds to the time from the commencement of the ventricular contraction to the moment when the organ has completed its change in shape from a flattened to a more rounded form. The highest part of the curve corresponds also in time with the opening of the semilunar valves as well as the firm closure of the auriculo-ventricular valves. The duration of this period in this case is only about 3-50ths of a second. The period from *c* to *d* is that during which the ventricle having grasped its contents is emptying its cavity and remaining contracted. It can be seen that only during the first half of this period is there any marked descent of the lever point; in other words, the antero-posterior diameter does not continue to diminish during the whole period of the systole, indicating that little or no blood was thrown out during the second half of this period, the ventricle remaining simply contracted after having emptied its cavity. The period from *d* to *a* is that during which the ventricular muscle is relaxing. Here, as is frequently the case, there is no period of pause between the close of the relaxation of the ventricle and the commencement of the succeeding distension. The tracing gives no evidence as to the time of closure of the semilunar valves.'—(*Text-Book of Physiology*, by Professor M. Foster, fourth edition, p. 147.)

valves, giving rise to the second sound, the ventricle has just finished its systole and is beginning to relax,' it would appear that during the commencement of the down-stroke the ventricles are still in systole.

The tracings of Chauveau and Marey, represented in fig. 304, afford, I think, some corroboration of this view. It will be seen by reference to the figure that the down-stroke of the third tracing, taken from the outside of the chest, is already considerably advanced before the sudden descent of the intra-ventricular pressure, which marks the relaxation of the ventricles (in the second tracing), occurs.

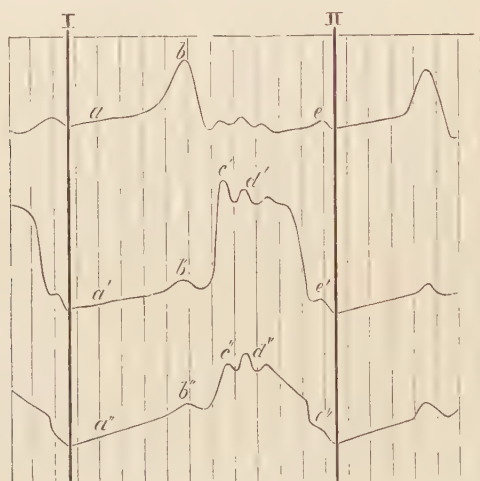


FIG. 304.—Simultaneous tracings, from the interior of the right auricle, from the interior of the right ventricle, and of the cardiac impulse in the horse. —(After Chauveau and Marey.) To be read from left to right.

The upper curve represents changes taking place within the auricle, the middle curve, changes within the ventricle. The lower curve represents the variations of pressure transmitted to a lever outside the chest and constituting the cardiac impulse. A complete cardiac cycle, beginning at the close of the ventricular systole, is comprised between the thick vertical lines I. and II. The thin vertical lines represent tenths of a second.

The line of ascent or up-stroke (a' to d , figs. 301 and 302) represents the impulse which is produced against the chest-wall by the rounding and hardening of the walls of the ven-

tricle which attend the commencement of the ventricular systole. The contraction of the ventricle, therefore, commences suddenly.

The commencement of the up-stroke slightly precedes the first sound of the heart which occurs during the ascent of the lever (a fact which is determined by listening to the heart sounds and watching the exact position of the lever of the cardiograph when the first sound occurs), and which reaches its maximum intensity as the lever approaches the summit of the up-stroke. The closure of the auriculo-ventricular valves (the sudden tension of which is, as we have previously seen, the main cause of the first sound of the heart) occurs, therefore, during the up-stroke. In some tracings the up-stroke is not quite straight, but is broken by a slight interruption or wave (*b*) (see fig. 305) which is supposed to indicate the closure of the auriculo-ventricular valves. This wave is only occasionally present.



FIG. 305.—*Cardiographic tracing from a case of aortic obstruction and regurgitation, with some mitral regurgitation, showing the wave b, which is supposed to indicate closure of the mitral valve.—(After Sansom.)*

The closure of the auriculo-ventricular valves ‘might be expected,’ says Dr Galabin, ‘to produce first, a slight check, and then an acceleration in the ascent; the check, due to the shock of the blood against the valves impelling the heart in the direction of its base; the acceleration, in consequence of the increased hardening of the ventricle as soon as it meets with resistance.’¹

The opening of the aortic valves corresponds to the summit of the up-stroke or line of ascent (*d*), and is separated,

¹ *Guy's Hospital Reports* 1875, p. 274.

therefore, from the commencement of the hardening of the walls of the ventricle by a distinct interval.¹

With the opening of the aortic valve there is, as we would expect, a fall of the lever corresponding to the relief which is afforded to the ventricle by the discharge of the first portion of its contents into the aorta. This fall is most marked when the ventricle contracts suddenly, and when the resistance to the opening of the valve is slight; *vice versâ* it is least marked when the ventricle contracts slowly, and when there is a considerable obstruction to the passage of the blood from the ventricle into the aorta. The obstruction may be situated at the aortic orifice (as in stenosis), or in the peripheral vessels (as in chronic Bright's disease). The apex wave is probably also in part due to the inertia of the instrument. In the tracing taken directly from the ventricle of the cat (see fig. 303) the descent of the lever, after the bursting open of the aortic valve, is seen to be very slight.

After the aortic valve has been opened, the ventricle, under normal circumstances, continues to contract for a considerable time. In tracings taken *directly* from the exterior of the ventricle (as in fig. 303), the period is probably represented, as Dr M. Foster points out, by the portion of the tracing which is included between the letters *c* and *d*. In tracings taken from the apex-impulse of man, the ventricular systole probably also includes (as has been previously pointed out) a portion of the subsequent down stroke (*d* to *g* in fig. 302.)

During the first part of this period (*i.e.* the part of the tracing which immediately follows *c* in fig. 303, and *d* in fig. 302), the blood is being expelled into the aorta; during the last part of the period (the part of the trace immediately preceding *d* in fig. 303, and *g* in fig. 302) the ventricle is empty, but still in a state of contraction.

A well marked wave (*f*) precedes the sudden descent of the lever in the normal cardiographic tracing from the apex

¹ The apex (*d*), of course, also represents the opening of the pulmonary valve, which event is, under normal circumstances, synchronous with the opening of the aortic segments.

beat of man (see figs. 301 and 302), but is not so well marked in the tracing taken directly from the ventricle of the cat. (See fig. 303.) Its exact significance is not definitely determined. Dr Galabin thinks that it is due to the locomotion of the heart as a whole.¹ Dr Sansom is of opinion that the wave represents forcible distention of the aorta at the end of the ventricular systole.²

Between the apex of the up-stroke (*d*) and the curve (*f*), a small wave (*e*) is sometimes seen. Observers are not agreed as to its exact significance ; possibly it may, I think, represent the resistance which the ventricle meets with (or rather the powerful contraction which is necessitated on the part of the ventricle) as it forces the blood into the arterial system, after the aortic valve has been opened.

The exact point in the tracing which marks the closure of the aortic valves is difficult to determine, and is not yet definitely settled. Professor M. Foster states with regard to this point, 'Hence we may infer, and the conclusion may be supported by other arguments, that at the actual closure of the semilunar valves, giving rise to the second sound, the ventricle has just finished its systole and is beginning to relax. If this view be correct, the time of the closure of the valves is not indicated on the cardiographic tracing by any special mark, but coincides with the commencement of the sudden fall of the lever,'³ as at *d* in fig. 303.

If we include a portion of the line of descent in the systolic portion of the tracing, the closure of the aortic valves should occur near the termination, rather than at the commencement, of the line of descent ; and in support of this view Dr Galabin states that under normal circumstances, and more especially when the arterial blood pressure is low, the second sound can

¹ In speaking of this wave Dr Galabin says, 'Thus we see that *f* corresponds in time to the maximum contraction of the ventricle. Since, however, when it takes the form of an elevation in the positive tracing, it cannot be due to the contraction of the ventricle as such ; its cause must be sought in some coincident occurrence, and this can only be the locomotion of the heart as a whole.'—(*Guy's Hospital Reports*, 1875, p. 270.)

² *Diagnosis of Diseases of the Heart*, p. 257.

³ *Text Book of Physiology*, fourth edition, p. 150.

be heard to occur towards the termination rather than at the commencement of this portion of the tracing. He concludes and, with his opinion, Dr Sansom also agrees, that the slight notch *g*, which is seen in some tracings (more especially when the arterial pressure is low), 'if not produced in the instrument may indicate the moment at which the valves close.'¹

When the blood pressure in the aorta is high, the second sound is heard to occur near the commencement, rather than near the termination of the line of descent, and it is not unreasonable to conclude that under such circumstances the aortic segments are closed before the relaxation of the ventricle has fairly commenced. In cases of this description (*i.e.* high arterial tension) the wave *g* is not seen in the tracing.

(The wave *g* does not, it must be observed, correspond to the waves *e*, *e'*, *e''*, in fig. 304, which, as Professor M. Foster very forcibly points out, can hardly, as has been supposed, represent the closure of the aortic valves.)

To sum up, then, the systolic portion of the cardiographic tracing of man, which commences at the point *a'* in fig. 302, probably includes a portion of the line of descent (*f* to *c*) and probably terminates somewhere about the point *g*; the closure of the aortic valves is probably represented by the notch *g* which occurs in some tracings, more especially when the arterial blood-pressure is low; the line of ascent (*a* to *d*) corresponds to the contraction and rounding of the ventricles; the apex (*d*) to the bursting open of the aortic valves; the wave (*b*) probably represents the closure of the auriculo-ventricular valves; the wave (*e*) possibly represents the resistance which the left ventricle meets with in propelling its blood, after the aortic valves have been opened, into the arterial system; the wave (*f*) is probably due to the movement of the heart as a whole, and probably represents the distention of the aorta which accompanies the emptying of the left ventricle.

The summit of the systolic portion of the tracing (*d* to *f*) is broad and sustained in hypertrophy; narrow and sharp in dilatation and in all conditions in which the contraction of

¹ *Guy's Hospital Reports*, 1875, p. 274.

the left ventricle is sharp, short and unsustained in conditions therefore of cardiac failure. (See figs. 306, 307 and 308.)

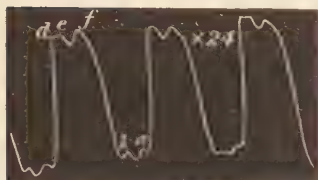


FIG. 306.

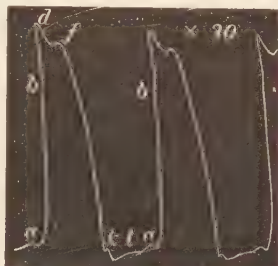


FIG. 307.

FIG. 247.—*Cardiographic tracing in hypertrophy of the left ventricle.* (After Galabin.)

‘Emily L., æt. 8. Loud systolic murmur at the apex, preceded by a very faint rumbling sound. A presystolic murmur has been heard previously. Heart much hypertrophied, P. 96.’ (*Guy’s Hospital Reports*, 1875, p. 313.)

FIG. 248.—*Cardiographic tracing in hypertrophy of the left ventricle.*—(After Galabin.)

‘Thomas G., æt. 56. Chronic Bright’s disease with atheromatous arteries. The cardiac impulse was very powerful, but no murmur was heard, P. 63.’—(*Guy’s Hospital Reports*, 1875, p. 312.)

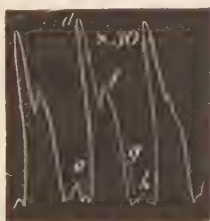


FIG. 308.—*Cardiogram in a case of exophthalmic goitre.*—(After Galabin.)

‘Rebecca S., æt. 20. Mitral regurgitation combined with exophthalmic goitre. Heart dilated and hypertrophied. Pulse 110.’—(*Guy’s Hospital Reports*, 1875, p. 314).

In addition, a series of irregular (serrated) curves is seen in the systolic portion of the tracing, in some cases in which rough systolic murmurs can be heard, or systolic thrills felt, over the præcordial region.

The diastolic portion of the tracing.

Under normal circumstances the diastole of the ventricle commences with, or more accurately just before, the closure of the aortic valves, which event is, as we have seen, probably represented in some tracings by the notch *g*. At the commencement of the ventricular diastole, the elastic recoil or expansion of the ventricle occurs, the mitral valve is burst open, and blood flows from the left auricle into the left ventricle; the auricular contraction or systole then occurs, and is almost immediately succeeded by the contraction of the ventricle.

Now, by observing the character of the diastolic portion of the tracing (the part numbered 2 in fig. 302) we obtain information as to the manner in which the ventricle is being filled, and since the filling of the left ventricle depends to a large extent upon the condition of the mitral orifice, we obtain, in some cases, information as to the condition of that valvular orifice. Further, we are able, in some cases, to note the character of the auricular contraction, and so to form a judgment as to the condition of the auricular muscle. If we grant that the closure of the aortic valve occurs towards the termination of the line of descent from *f* to *c*, the diastolic portion of the tracing may be said to be represented, under normal circumstances, by a slow and gradual ascent interrupted by two or three small waves (*k*, *l*, and *a*).

When the blood-flow into the ventricle is unusually rapid, as it is for instance in free regurgitation through the aortic or mitral orifices, and more especially when both aortic and mitral regurgitation are present, the line of ascent which marks the diastolic filling of the ventricles is much more abrupt than usually. (See figs. 309 and 310.)

When, on the contrary, the blood-flow into the ventricle is abnormally slow, as it is in mitral stenosis, the diastolic portion of the tracing may be of much longer duration than in health.¹ (See fig. 311.)

¹ I say *may* be, for in mitral stenosis the rhythm is often perverted; the left auricle is apt to contract at irregular intervals, and in some of the individual cardiographic curves the diastolic portion may be shortened, while in the majority it is, as we should expect, increased.

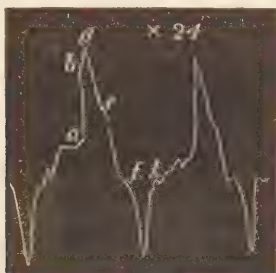


FIG. 309.—Cardiogram in a case of aortic regurgitation.—(After Galabin.)

‘The heart was much dilated, the apex beat being in the sixth intercostal space, and external to the line of the nipple. The pulse tracing showed extreme collapse in the diastolic portion, and an almost entire absence of the tidal wave. From this it may be inferred that the regurgitation was very free, and the contractions of the heart short and incomplete. The tracing is partly inverted, and a retraction occurs during the latter part of systole, followed by a sudden recoil.’
—(*Guy's Hospital Reports*, 1875, p. 312.)



FIG. 310.—Cardiogram from a case of aortic regurgitation and obstruction combined with mitral regurgitation showing marked ascent of the line indicating intra-ventricular pressure during diastole.—(After Sansom.)



FIG. 311.—Cardiographic tracing in a case of mitral stenosis.—(After Galabin.)

‘Matilda A., æt. 37. Long, rough, presystolic murmur, commencing immediately from the second sound, and leading up to the first sound. Pulse 57.’
—(*Guy's Hospital Reports*, 1875, p. 314.)

The *duration* of the diastolic portion of the tracing indicates the length of the ventricular diastole; the mode of *ascent*, the manner in which the ventricle is being filled.

The presence of rough presystolic or diastolic mitral murmurs and thrills, is in some cases manifested in the tracing by a series of fine serrated curves, which occasionally also mark the presence of a rough diastolic aortic murmur.¹ (See figs. 312 and 313.)

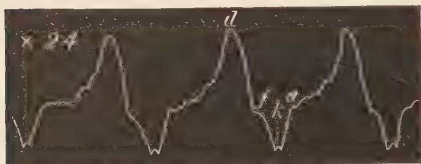


FIG. 312.

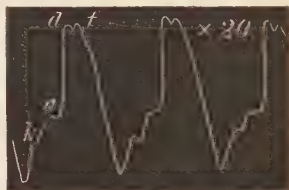


FIG. 313.

FIG. 312.—*Cardiographic tracing in a case of mitral stenosis.*—(After Galabin.)

‘George M., æt. 19. Long, loud, and harsh presystolic murmur, commencing immediately from the second sound and running up to the first sound. Pulse 60. The letter *a*, indicates the probable commencement of the auricular contraction.’—(Guy’s *Hospital Reports*, 1875, p. 314.)

FIG. 313.—*Cardiogram from a case of aortic regurgitation.*—(After Galabin.)

‘Thomas S., æt. 45. The diastolic murmur was very loud and accompanied by a thrill felt at the apex, P. 74.’—(Guy’s *Hospital Reports*, 1875, p. 313.)

The exact significance of the wave *k* (see fig. 302), which occurs near the commencement of the diastolic portion of the tracing is a matter of dispute. Dr Galabin thinks that it probably indicates a slight movement forward of the heart as a whole caused by the reflux of blood which closes the aortic valve.² Dr Sansom has found the wave *k* to be most constantly associated with *suddenness* in the action of the ventricles, and he states that suddenness of the action of the ventricles, and especially of the diastolic relaxation, appears therefore to be the probable cause of its high development.³

When the blood-flow into the auricle is rapid, the wave *k* may not be perceptible, but may be merged with the rapid rise which is present in the whole of the diastolic portion of the tracing. ‘In some cases of very free regurgitation with, at the same time, great hypertrophy, there is not,’

¹ The vibrations of diastolic aortic murmurs are seldom sufficiently rough to be demonstrated in this manner.

² Guy’s *Hospital Reports*, 1875, p. 276.

³ *Diagnosis of Diseases of the Heart*, p. 262.

according to Dr Galabin, 'a gradual ascent leading up to the systole, but a very marked rise followed by a fall. It seems that the aortic pressure being raised to a very high point in systole, causes the blood to flow back into the ventricle with a powerful momentum during diastole, which produces its effect on the trace, not only by raising the ventricular pressure, but by impelling the heart bodily against the ribs.'¹ In one case of this description, observed by Dr Galabin, the impulse corresponding to the wave *k* was so great, as actually to be mistaken for the apex-beat produced by the ventricular contraction itself.

The wave *l* seems to correspond to the blood-flow from the auricle into the ventricle, or the 'passive venous flow,' as it has been termed, to distinguish it from the blood current produced by the *contraction* of the muscular wall of the auricle.

The wave (*a*) which is seen in many normal tracings, but only when the apex-beat is well defined and the tracing a good one, is acknowledged by all authorities to represent the contraction of the auricle. When this wave (*a*) is well marked, we may infer that the muscular wall of the left auricle is contracting forcibly. The height and breadth of the wave (*a*) are indications of the force of the auricular contraction, *provided that there is no obstruction at the mitral orifice*. Its exaggeration is, in fact, in many cases an indication of hypertrophy of the left auricle. Now hypertrophy of the left auricle may occur both in mitral regurgitation and in mitral stenosis. In the former case (mitral regurgitation) the duration of the ventricular diastole is shortened, and the diastolic portion of the trace is represented by a rapid ascent; in some cases of this description the wave *a* is merged in the general rapid ascent, in others it is differentiated and distinct. In the latter (mitral stenosis), the diastolic portion of the tracing is unduly prolonged, and if the stenosis is considerable, even although the muscular wall of the left auricle is hypertrophied, the wave *a* may not be exaggerated, in fact, it may not be present, the obstruction of the mitral orifice preventing, as it were, the force of the auricular contraction

¹ *Guy's Hospital Reports*, 1875, p. 279.

being communicated to the recording lever of the cardiograph. In other cases of mitral stenosis in which the obstruction is not very great, and in which the wall of the left auricle is hypertrophied, the wave (*a*) is exaggerated. In others, it (*a*) does not immediately precede the ventricular systole, but occurs in the earlier part of the diastolic portion of the tracing, the auricular contraction occurring earlier in the diastole than normal. In exceptional circumstances the rhythm of the auricular contractions may, it would appear, be still further interfered with, such at all events seems to be the most probable explanation of a cardiogram published by Galabin (see fig. 314); in that case, which was probably,

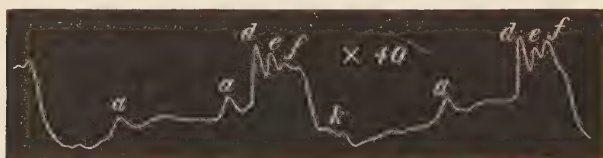


FIG. 314.—Cardiogram from a case of (?) mitral stenosis, showing two auricular contractions (*a*, *a*₁) for one ventricular beat. —(After Galabin.)

though not certainly, a case of mitral stenosis, the diastolic portion of the trace was enormously prolonged, and in many of the cardiographic curves two auricular contractions appeared to be present; in other words, each auricular contraction was not, as under ordinary circumstances, followed by a contraction of the ventricle.

The special characters of the cardiographic tracing in the individual valvular lesions have previously been described, and need not again be detailed.

Inverted tracings.—In describing the manner in which the cardiograph is to be applied, it has been stated that unless the button of the instrument is very accurately adjusted to the maximum point of pulsation of the apex-beat, the tracing is apt to be inverted; beyond a certain area in fact the (positive) impulse of the heart against the chest wall is replaced by (negative) suction or retraction, and when the button of the cardiograph is placed over the area of retrac-

tion, a negative or inverted tracing is obtained. Between the (positive) area of impulse and the (negative) area of retraction, there is, in many cases, an intermediate area which yields a mixed tracing (partly positive and partly negative.)

Inverted tracings may be deciphered by means of a mirror; or may be read as positive if they are turned upside down and read from right to left.

Completely inverted tracings are of some value, but no importance should be attached to mixed tracings (partly positive and partly negative).

The following cardiograms, copied from Galabin, are good illustrations of negative tracings. (See figs. 315, 316, and 317.)

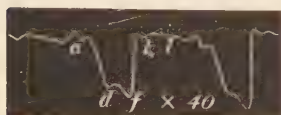


FIG. 315.



FIG. 316.

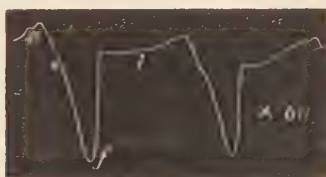


FIG. 317.

FIGS. 315, 316 and 317.—*Inverted tracings.*—(After Galabin.)

‘Inverted tracings taken in different situations from a healthy heart which gave no positive impulse anywhere. Fig. 315 was taken near the usual position of the apex beat; fig. 316 in the epigastrium, to the left of the middle line; fig. 317, also in the epigastrium, but to the right of the middle line.’

For further information on the cardiograph, the reader is referred to the writings of Foster,¹ Galabin,² and Sansom,³ to which I am largely indebted for much of my information on the subject.

¹ *A Text Book of Physiology*, by Professor M. Foster, p. 138, *et seq.*

² *On the Interpretation of Cardiographic Tracings* by Dr A. L. Galabin *Guy's Hospital Reports*, 1875, p. 261.

³ *Diagnosis of Diseases of the Heart*, by Dr Sansom, p. 221.

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